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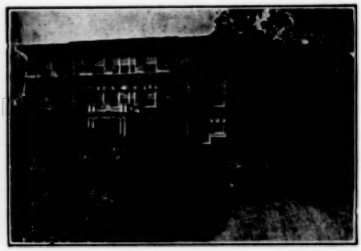

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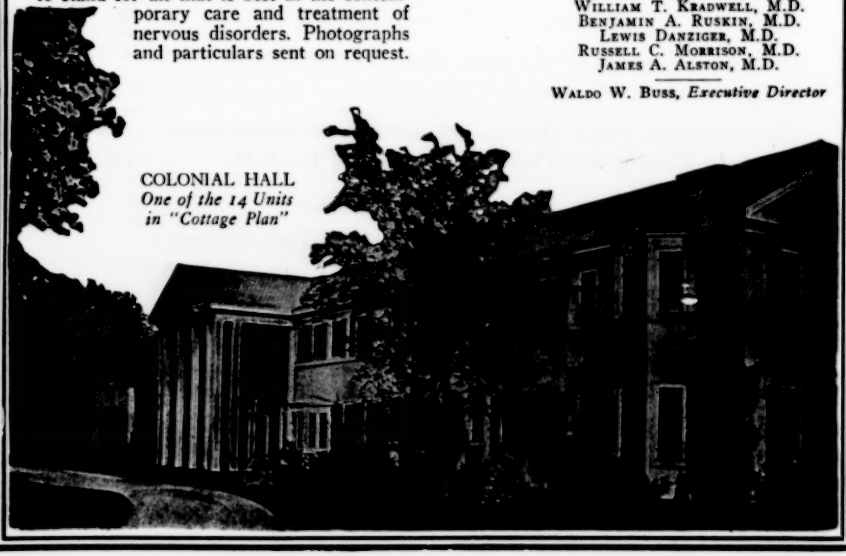

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The Journal of Nervous and Mental Disease

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DEMENTIA INFANTILIS

WILFRED C. HULSE *

Introductory Remarks

In recent years, psychotic states in young children have been frequently described in American literature. Their relationship to schizophrenia is still a matter of debate although the term "childhood schizophrenia" has come into wide use. The basic articles by Howard Potter, Louise Despert, Leo Kanner, Loretta Bender, Margaret Mahler and other American contributors are well known to all interested in this subject, but earlier European literature on psychosis, schizophrenia and dementia praecox in childhood is hardly accessible to the American reader.

Occasionally authors in this country make use of the diagnostic term, "Heller's syndrome," as a disease sui generis and different from infantile autism, pseudofeeblemindedness or childhood schizophrenia. It seemed advisable to make available an English translation of one of the original papers by Theodor Heller which describes his "syndrome" and tries to differentiate it from other psychotic or schizophrenia-like illnesses in childhood. Whether such a clinical differentiation is valid remains for the reader to decide.

The paper is here rendered in its entirety in as literal a translation as possible. Some overly long German sentences have been broken down into several parts. To avoid semantic confusion, a short glossary of German and English technical terms has been added. Heller's

* Child Psychiatric Section, The Mount Sinai Hospital, New York.

paper appeared originally in the 5th (Schluss-) Heft, 37. Band, pages 661-667, *Zeitschrift fuer Kinderforschung*, Berlin: Julius Springer Verlag, 1930. (It has been translated through their kind permission.) Theodor Heller was a collaborating editor of that journal.

About Dementia Infantilis

BY

THEODOR HELLER

At the Fifth Congress for Remedial Education at Cologne (1930) Professor Corberi of Milan submitted a discussion remark in Italian following the lectures by Professor H. W. Maier (Zuerich) and by Dr. P. Seelig (Berlin) concerning schizophrenia-like conditions in childhood. Lack of time prevented a translation and reading of Professor Corberi's remarks at that meeting. These remarks deal primarily with dementia infantilis which had been observed and described by me for the first time. Professor Corberi compares dementia infantilis with the dementia praecocissima described by Sante de Sanctis. He concludes that these two symptom-complexes which often are confused with each other and are erroneously taken to be identical represent actually two different conditions. The assumption that dementia praecocissima is an early form of dementia praecox in childhood is also incorrect. It is not easy to gain a completely clear understanding from the available literature about Sante de Sanctis' concept of dementia praecocissima as he seems to include under this designation different types of psychoneuroses of early childhood, all resulting in idiotic regression. I have asked Professor Corberi who has published on this subject† a number of times to clarify by means of a detailed summary what Sante de Sanctis and his school understand by the term "dementia praecocissima." We will then be able to judge the validity of objections made by some German researchers who believe that in dementia praecocissima we are dealing primarily with postencephalitic states, progressive forms of mental deficiency and similar cases. Yet such revisions and critiques have to be reserved for the medical author. I will restrict myself to giving in the following pages a picture of dementia infantilis to the extent to which my research and my experiences have enabled me to gain insight into this condition and its interrelated factors.

During the years 1905 and 1906 I was given to observe and to evaluate an unusually large number of feebleminded children. I was particularly impressed by a number of cases whose past histories were so similar that there could remain no doubt as to a close inner connection. These children came from different countries and from differing social strata. These were children who—without preceding illness—had become conspicuous in

† Corberi: Una sindrome demenziale grave nell' età infantile. *Atti della Società Lombarda di Scienze Mediche e Biologiche*. Vol. XIII.—Fasc. VI. Milano, 1925.

Ibid: Sindromi di regressione mentale infanto-giovanile. *Rivista di Patologia nervosa e mentale*. Vol. XXXI. Fasc. 1. Firenze. Gennaio-Febbraio, 1926.

their third and fourth years through early symptoms which one might sum up (if such a designation is appropriate at this early age) as character changes which presented themselves primarily by changes of mood. These had been placid or lively children by nature until that time; now they became moody, negativistic, disobedient, often raging without reason, and whining; not rarely they started to destroy their toys of which they had made reasonable use before. Many showed definite anxiety states, occasionally of a hallucinatory character. With these stormy symptoms, or in rare cases more quietly and without affective blows, there developed a mental process of regression which led within a few months to a complete loss of speech and to complete idiocy. The language became impoverished by degrees, words were distorted, sentences could not be repeated any longer, and finally, the children stopped speaking altogether. The capacity to understand language was lost with the exception of some primitive remnants. During this regressive process, motoric degeneration became frequently evident. The children acquired tic-like movements; they grimaced, posed in peculiar positions. Their condition reminded one of the erethism of certain idiots. Many became incontinent, losing control of urine, sometimes also of feces; it became necessary to feed them. During all this there remained a certain degree of attentiveness which was particularly evident in their ability to focus ("Blickrichtung").

Without exception, all parents maintained that the children had been intelligent before the onset of this condition. In a few cases, the information given by the parents suggested that some of these children had been very gifted. Of course, we had to be cautious in evaluating these statements, as it is well known that parents generally overestimate the intellectual achievements of their children, especially in retrospect. But there was no doubt that these children had once been able to speak and to understand the spoken word, that they had possessed the ability for independent action commensurate to their ages. In no way could they have been called very retarded or feebleminded, nor had their character development been particularly conspicuous.

The lowest mental level was reached within an average of nine months. No improvement was observed in any of the children after that. As a rule the children remained from then on in a state of extreme idiotic regression. Great motor restlessness, stereotypies and tic-like habits remained in the foreground. Throughout this, the children maintained their misleading intelligent facial expression. Particularly impressive was the clear look in their eyes and the apparent attention which these children frequently seemed to pay to whatever went on around them. They proved to be unresponsive to any approach through remedial education. Through careful therapeutic nursing some could be trained to eat by themselves and to keep clean.

As a rule the parents expected a recovery of the lost functions; they were cruelly disappointed as the low mental status persisted and as the child lapsed into complete idiotic regression.

In 1908 I published 6 cases in the Journal for Research and Treatment of Juvenile Feeblemindedness (*Zeitschrift fuer die Erforschung und Behandlung des jugendlichen Schwachsinns.* Jena: Gustav Fischer). I had illustrated this article with photographs of these children. During his stay at Vienna in 1907, Professor Weygandt had examined psychiatrically one of my cases. I had previously acquainted him with my observations and had suggested the designation dementia infantilis for these cases. Professor Weygandt was able to concur completely with my observations and also accepted the name dementia infantilis. This term was chosen merely to designate a state of mental deterioration occurring in childhood; it did not intend to indicate in a more precise manner the origin of this process nor to connect it with other symptom complexes of similar nature. Prof. Weygandt published the results of these observations during the same year in the above mentioned journal under the title "Idiotie und Dementia praecox." Through an oversight the paper fails to refer to my observations and communications.

Various authors have since described cases of dementia infantilis (Higier, Jancke, and others). More recently in 1921, Julius Zappert made dementia infantilis the topic for a presentation at the meeting of the German Pediatric Association (*Deutsche Gesellschaft fuer Kinderheilkunde*) at Jena. His material comprised 13 cases, in all of whom he could consistently observe the following stages:

1. Normal mental and physical development during the first years of life;
2. Onset of the illness between the third and fourth years of life;
3. Psychic and intellectual changes demonstrated by ineffectiveness of educational and recreational (zerstreuender) influences; marked restlessness, excited and occasionally anxious behavior, increasing dementia;
4. Appearance of speech disorders in the beginning and during the course of the illness;
5. Maintenance of motor functions and complete lack of focal symptoms from the central nervous system;
6. Final complete idiotic regression;
7. Non-imbecilic facial expression and looks ("Blick").

I have collected case material of 28 children with dementia infantilis. All of them had thorough medical examinations so that any confusion with other diseases may be completely ruled out. Special caution was taken to prevent the sneaking-in of encephalitis lethargica cases. Any case where doubts existed about the parental reports, even if they described a regressive process similar to dementia infantilis, was excluded. Likewise, cases were not included in the above number if no definite record could be established because of a preceding illness. In all cases, the catastrophic mental decline occurred in the same fashion, the past histories conformed in all important features, if one discounted the self-deceptive stories of par-

ents reporting short-lived temporary improvements. I have nothing further of importance to add to my observations published in 1908.

The reports about the follow-up studies of children who have gone through a dementia infantilis are of interest. All of the children remained in complete idiotic regression, did not speak, did not understand anything or only very little. They were unable to keep themselves occupied, were restless and years later still showed tics and sterotyped movements similar to those which had developed during or immediately after the (acute state of the) illness. The majority had been referred to nursing homes or to mental institutions, as they could not be kept at home because of their restlessness. Malaria therapy was tried in one case but no success was noted after repeated attacks of fever. Some of the patients had by now reached adult age; despite their severe idiotic regression, they gave the superficial impression of much greater intelligence than the idiots with whom they were placed. Their looks and the expression in their eyes ("Blick") encouraged many of the unfortunate parents not to give up hope for recovery, even after years of a continuous low mental level. Again and again physicians and directors of institutions were approached by these parents and asked to initiate "treatment" ("Kuren"). In one instance, parents had traveled from one therapist to the next having been told about someone's efforts to cure feeblemindedness. Finally, they began to consult quacks of different schools and vocations. After all these many trials and tribulations, having tried every possible thing, the condition of the patient had at the very end not improved in the slightest noticeable degree.

Professor Emil Redlich to whom I was able to present several of these ill children, concerned himself with the question whether we might not possibly be dealing with an early form of dementia praecox. At first he thought that there seemed to exist a certain similarity between dementia infantilis and some forms of schizophrenia. Later he was amazed to find the nearly photographic likeness of the course the illness took. He then became convinced that dementia infantilis was a peculiar regressive process which did not belong to the group of schizophrenias, which was of an unknown nature, impossible to speculate about.

Professor Maier and Dr. Seelig have pointed out in the above-mentioned lectures that cases belonging to the schizophrenias are extremely rare in childhood and might not exist at all. During a period of 35 years I have had the opportunity to examine and evaluate an extremely large number of abnormal children. In all this time I only once had the occasion to observe a case of schizophrenia in childhood, which has been established beyond any doubt. This is the case of S.S., born March 24, 1917. From her past record we learned the following: She is the second child of healthy Jewish parents. No cases of mental disorders or nervous diseases have occurred in this family. Her birth was normal and so was her development during the first years of life. She learned to walk and to speak at the normal time. She showed only feeding difficulties—she belonged to the group of poor eaters. There was no other educational unusualness; she was intelli-

gent, liked picture books and story telling. She understood all she was told and asked reasonable questions. This her father was able to show by many convincing examples. She began to show peculiarities at the age of five, laughing occasionally without reason, whispering to herself words which made no sense. Later she showed a preference for odd positions, appeared sometimes as though "rooted to the spot," would get stuck occasionally in the middle of an action, for instance, raising her hand with a spoon without getting it into her mouth. It also happened that she would suddenly accelerate her steps and start to run. She ran away from her nurse during outdoor walks. She lost interest in games, became whining, negativistic, incontinent. At the age of seven, one could not induce her to get up; she stayed in bed and had to be spoon-fed, taking little nourishment, often for several days. She lost control over urine and feces and remained lying in this dirty mess. On Dec. 5, 1924 she was brought to Vienna and was admitted to my institution. She was first examined by Professor von Wagner-Jauregg who stated that we were dealing with an early form of dementia praecox, a diagnosis which was confirmed in every way during the subsequent period. The child was first catatonic with outspoken flexibilitas cerea. Occasionally, this catatonic state was interrupted by a state of excitement. In this state, the child would run around, touch everything, throw things to the floor as if in a state of high spirits. Everything edible was consumed ravenously by S.; her voracity had no limits at times like this. She spoke Polish and German words in a disconnected way. She carried out orders; on occasions she even did some sewing or other little housework during such a period showing remarkable manual dexterity. Then suddenly and unexpectedly the catatonic state returned, the child sat in her little chair in frozen poses, she had to be fed and would use the toilet only when ordered and with adult help. But one had the impression that even in the catatonic state, S. observed whatever was going on around her. On occasions one could find proof of this: when once again, she moved freely about the room, she found without hesitation things in the closet which the nurse had put there while the child had been lying in bed, apparently completely withdrawn. Once while being left alone in bed in a frozen pose, she got up, ate within an extremely short time the nurse's supper that was left on the table in the bedroom and was found again motionless in bed by the returning nurse.

Step by step, the child's intelligence declined more and more. She stopped speaking, became unable to keep herself occupied in the simplest fashion, and was unable to understand the meaning and use of the tools and toys put at her disposal. The catatonia moved into the background more and more and was replaced by a state of silliness and idiotic regression. The child now sits idly in her little chair, laughs without cause, plays monotonously in the manner of idiots with little blocks or plates and displays a ravenous appetite which she tries to satisfy with garbage, left-overs and crumbs from the floor. Teaching attempts find the child without response and in complete apathy. She does not take notice of other children and

shows no interest in their games and occupations. She lately gained a lot of weight. She is just approaching the onset of puberty.

No further explanation is needed to show that this early form (Frueh-form) of schizophrenia is entirely different in its course from the cases of dementia infantilis which have been observed until now. Cases of encephalitis lethargica are also sharply differentiated from dementia infantilis. Children with encephalitis present a picture totally different from our cases. Besides, catastrophic declines of intelligence which we have learned to recognize as absolutely typical for dementia infantilis, do not occur in patients with encephalitis. Complete idiotic regressions can probably be observed but rarely in encephalitis lethargica.

Physicians and educators depend on one another for the diagnosis and treatment of feebleminded juveniles; they depend on mutual help and mutual co-operation. This has been the way in which all progress has been achieved in orthopedagogics. Whenever the remedial educator (orthopedagogist) makes observations in his practice which can contribute to medical research, he should not hesitate to communicate such material. In the same manner, the physician should not miss any opportunity to acquaint the educator with facts, experiences and observations which can stimulate and enrich the practical fields of teaching and education. In the area of remedial education, the arts of medicine and pedagogy interlink with each other; they also overlap in part and it is just through this common cultivation of certain areas, that the best and most persistent successes have been achieved. Many areas in the science of infantile feeblemindedness are still very much in the dark. Enlightenment is needed for a better concept and a clearer understanding of the therapy and its limitations. This is how I should like to have understood the goals and purposes of my observations which the remedial educator offers to the physician.

GLOSSARY

Blows of affect	Affektstoesse
Catastrophic mental decline	geistiger Absturz
Frozen poses	starre Posen
High spirits	Uebermut
Idiotic regression—idiocy	Verbloedung
Mental deterioration	geistige Veroedung
Motoric degeneration	Entartung der Motorik
Remedial Education (Orthopedagogics)	Heilpaedagogik
Symptom complexes	Krankheitsbilder oder—erscheinungen
Therapeutic nursing	Pflegebehandlung

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ELECTROCONVULSIVE THERAPY IN THE PRESENCE OF PHYSICAL DEFECTS

WILLIAM K. McKNIGHT, M.D.,
RALPH BURBRIDGE, M.D.,
AND
PAUL GUTH, M.D.*

In 1938, Cerletti introduced the use of electrically induced convulsions for therapeutic purposes in man (5). Because of the well known dangers associated with electrical currents, there was widespread reluctance to accept this new therapeutic modality. A preliminary paper submitted by Barrera and Kalinowsky was returned by the editor with a note saying, "There is little doubt but that the passage of currents of 300 milliamperes through the brain would be a highly dangerous procedure and in all probability result in fatality to the patient" (26). Time soon showed these fears to be unfounded. Physically well persons showed few adverse effects following electricconvulsive therapy. Physically ill patients, however, continued to be a cause of considerable concern. Numerous physical contraindications to convulsive therapy were listed initially (cerebral lesions, tuberculosis, advanced heart disease, glaucoma, etc.) (22). Nevertheless, there was early recognition (1942) of the importance of considering each patient individually and balancing the advantages to be gained by a possible psychiatric recovery against the additional risks imposed by the existing physical conditions (23). As experience has broadened, the list of definite contraindications has dwindled markedly (i.e., congestive heart failure, recent myocardial infarction, and aneurysm of the aorta) (26), and this has borne out the importance of the preceding statement. Kalinowsky has said "preexisting physical disease should not be overstressed as contraindicative of a treatment, which, although admittedly drastic, results in few ill-effects on the organism as a whole" (13).

Since the first use of electroconvulsive therapy at this hospital in 1939, the question of physical contraindications to this therapy has arisen frequently at Staff Conference, as it has elsewhere. A review of this experience is the subject of the present paper and the cases cited were studied during the seven year period from February 1946 to January 1953 during which approximately 23,520 electroconvulsive treatments were given.

* Friends' Hospital, Philadelphia 24, Penna.

Method of Treatment

From January 1946 to May 1950, the Cerletti-Bini machine was used, and, from May 1950 to the present, the Medcraft machine with glissando. (The Reiter machine was tried for one year—December 1947 to December 1948—but was discontinued because of pre-treatment apprehension which, without medication, was considered to be excessive.) Patients are routinely given curare before treatment (1.0 cc. per 40 lbs. of body weight) with a maximum dosage of 3 cc. unless a contraindication (i.e., sensitivity) is present. In most instances this dose does not produce more than a slight heaviness of the eyelids. Usually, electroconvulsive therapy was given three times a week, the duration of treatment varying with individual cases.

Case Analysis

During the seven year period of this report, 124 patients (43 males and 81 females) presented physical ailments of sufficient severity to warrant special consideration by the staff before electroconvulsive therapy was ordered.

Age (Table I). The cases studied covered the entire age range from early adulthood to old age, and no significant conclusions were drawn from the age breakdown, except for results in the older age group which will be discussed later.

TABLE I.—Age Incidence

	15–25	*26–35*	*36–45*	*46–55*	*56–65*	*66–75*	*76–85*	*Totals*
Male	1	5	1	11	17	6	2	43
Female	5	9	13	19	20	12	3	81
Totals	6	14	14	30	37	18	5	124

Psychiatric Diagnoses (Table II). As this is not of primary importance to this report only large diagnostic groups are included. The cases studied covered a wide range of psychiatric disorders but no pertinent conclusions were drawn from this breakdown.

TABLE II.—Diagnostic Incidence

Diagnostic List	*Male*	*Female*	*Total*
Involutional psychoses			
a. Melancholia	12	28	40
b. Paranoid	1	7	8
c. Mixed (a & b)		1	1
d. Total			49
Manic-depressive psychosis			
a. Manic type	1	1	2
b. Depressive type	7	9	16
c. Total			18

TABLE II.—*Continued*

Diagnositic List	*Male*	*Female*	*Total*
Schizophrenia			
a. Catatonic type	1	3	4
b. Paranoid type	6	12	18
c. Type not listed	1	4	5
d. Total			27
Psychoneurosis			
a. Unclassified (usually mixed with depression)	2	2	4
Paranoid condition	2	0	2
Reactive depression	6	7	13
Psychosis with cerebral arteriosclerosis	2	2	4
Alzeiheimer's disease	1	1	2
Psychotic depression	1	3	4
Psychosis with organic nervous disease probably pernicious anemia	0	1	1
TOTAL	43	81	124

Analysis of Physical Factors. The various physical complicating factors are listed in Table III as follows:

TABLE III.—COMPLICATION INCIDENCE

Complicating Factors	*Male*	*Female*	*Total*
Cardiovascular system			
Hypertension	10	10	20
Hypertensive heart disease	5	4	9
Arteriosclerotic heart disease	5	5	10
Rheumatic heart disease	0	9	9
Heart disease	0	1	1
Age	2	8	10
Circulatory collapse	1	0	1*
Cerebral vascular accident	2	1	3
Cardiac arrest during previous ECT	1	0	1
Hemopoietic system			
Polycythemia	1	0	1
Bleeding tendency	0	1	1
Respiratory System			
Pulmonary tuberculosis	2	0	2
Pneumoconiosis	2	0	2
Bronchiectasis	1	0	1
Hemoptysis	2	0	2
Bronchial asthma	1	1	2
Emphysema	0	2	2
Osseous system			
Hemilaminectomy	1	0	1
Old vertebral fractures	0	4	4
Vertebral column changes (arthritis, etc.)	0	3	3
Arthritis, generalized, rheumatoid	0	2	2
Hip fracture (1 old, 1 recent)	0	2	2
Breast cancer	0	1	1
Leg amputation	0	1	1
Childhood rickets and achondroplasia	0	1	1

* Autopsy—pulmonary infarct. & congestion.

TABLE III.—*Continued*

Complicating Factors	*Male*	*Female*	*Total*
Nervous system			
Old anterior poliomyelitis	0	2	2
Neurological disease posteriorlateral sclerosis	1	3	4
Multiple sclerosis	1	1	2
Undiagnosed disease of CNS (with cerebral atrophy on pneumoencephalogram)	0	1	1
Parkinsonism	2	0	2
Friedrich's ataxia	0	1	1
Encephalitis	0	1	1
Postencephalitic psychosis	1	0	1
Postelectroconvulsive therapy confusion and memory loss	0	1	1
Syncope	0	4	4
Special senses			
Otosclerosis	0	1	1
Conical cornea	1	0	1
Detached retina	0	1	1
Metabolic			
Diabetes mellitus	3	3	6
Thyrotoxicosis	0	2	2
Malnutrition with acetonuria	0	1	1
Gastro-intestinal			
Gastric ulcer, duodenal ulcer with gastrectomy	1	1	2
Inguinal hernia	0	1	1
Infections			
Low grade fever	0	1	1†
Soft tissue injury	0	1	1
Hematuria, cause undetermined	1	0	1
TOTAL	42	87	129

† Died after 2 ECT Autopsy—Bilat. L.L. Pneumonia.

1. Old Age: Sixteen patients (6 males and 10 females) over the age of 70 received electroconvulsive treatment, all showing evidences of generalized arteriosclerosis and 10 presented additional physical disorders including arteriosclerotic and hypertensive heart disease (including one old myocardial infarction), mild hypertension and parkinsonism. All received a course of five to twelve treatments and 2 patients (including an 84-year-old woman with a hypertensive cardiovascular disease and a blood pressure of 220/110) were carried on maintainence electroconvulsive treatments (one treatment approximately every two weeks, as needed), receiving over 25 treatments. (In all instances, treatment was given for affective disorders and not for other [degenerative] symptoms of arteriosclerotic brain disease alone.) In one patient, therapy had to be stopped after five treatments because of marked confusion. No other patient showed any adverse physical effects from electroconvulsive therapy. Our findings indicate that old age by itself is not necessarily a contraindication to electroconvulsive therapy. This con-

firms the findings of others (9, 16, 24). It can be noted here that Lovell (16) suggests two instead of three treatments a week as there is a greater tendency for post-shock confusion in the aged, and Wilbur (24) suggests curarization to modify the convulsions.

2. Diseases of the Cardiovascular System: The majority of our possible physical "contraindications" fell in this category. The ill effects of excessive bodily activity upon a damaged myocardium and/or vascular system are well known. The marked muscular activity associated with a convulsion caused many to question the advisability of giving electroconvulsive therapy in the presence of such diseases. Clinical experience here and elsewhere has shown these fears to be not entirely warranted.

Coronary artery disease (previous myocardial infarction, angina pectoris, abnormal electrocardiogram manifestations) has caused most concern. Our series included 3 cases with electrocardiographic evidence of old myocardial infarctions (one or more years old), 3 with possible old myocardial infarction (history but no residual electrocardiogram evidence), and 2 with angina pectoris (with electrocardiographic evidence of coronary artery disease). All patients were in their 50's, except one aged 67 and two who were in their 70's. These patients each received one course of electroconvulsive therapy, varying from 5 to fourteen shocks, treatment being given every other day. No physical difficulties were encountered in any of these patients, and all responded well mentally. However, one other patient, a 69-year-old woman with arteriosclerotic heart disease, developed a myocardial infarction immediately following her sixth electroconvulsive therapy. She recovered from this attack but four months later she died at home from a second heart attack. Another patient with a previous myocardial infarction was refused electroconvulsive therapy, and, while on a course of benzedrine—15 mg. daily (for depression)—developed another infarction. The strong possibility of these patients having an infarction even when given no stimulant therapy cannot be ruled out, especially in view of the marked hyperactivity of the agitated depressed state in many such patients. These experiences point up the admitted dangers of electroconvulsive therapy, and warn against the indiscriminate use of this modality in patients with cardiovascular disease where the psychiatric illness does not definitely warrant the risk. Cardiac status, age, etc., should be carefully considered together.

There were 10 patients with arteriosclerotic heart disease (5 men and 5 women), including 3 with previous coronary occlusion and 2 with angina pectoris, mentioned above. From four to ten electroconvulsive treatments were given to these patients (three per week) with no com-

plications other than the myocardial infarction mentioned above. Nine patients with chronic rheumatic heart disease (all women), including one in marked decompensation who had to receive cardiac therapy (digitalis, ammonium chloride, and mercurial diuretics), before electroconvulsive therapy could be started, received 10 to 39 electroconvulsive treatments without any complications developing. Nine patients (5 men and 4 women) had hypertensive heart disease with blood pressures of 220/110 to 250/150, and electrocardiographic evidence of left ventricular strain. One patient, discussed above, had a previous coronary occlusion. All of this group were compensated and from 6 to 36 electroconvulsive treatments were given with no complications developing. Hypertension alone was present in 20 patients (10 males and 10 females), with blood pressures of 165/110 to 228/120. From two to 20 electroconvulsive treatments were given with a complication developing in only one. This patient, a 57-year-old male, involutional melancholia with a blood pressure of 228/120, responded well to two electroconvulsive treatments, but refused the third treatment and became so panicky that the treatment was not given. One day later (three days after the last electroconvulsive treatment) he developed a mild hemiparesis from which he recovered with minimal residua in one month. The severe emotional stress related to the refusal of treatment may have played a greater role in the pathogenesis of this cerebrovascular accident than had the two preceding electroconvulsive treatments.

Another cardiac case presented an unusual picture. A 65-year-old male, involutional melancholic, had developed cardiac arrest on two successive electroconvulsive treatments, eight months prior to his present admission here. He showed no clinical cardiac damage and a normal electrocardiogram. On the basis of a re-evaluation a medical consultant recommended giving atropine sulfate, grains 1/50, 20 minutes prior to each treatment, and he was given seven electroconvulsive treatments with no physical complications and he improved markedly in his mental state.

Thus, of 48 patients with cardiovascular disease sufficiently severe to warrant concern in prescribing electroconvulsive therapy, but who nevertheless were given electroconvulsive therapy, complications developed in only 2 patients, and one complication was probably more closely related pathogenetically to the patient's agitated emotional state than to the electroconvulsive therapy.

These findings are in accord with previous reports of clinically manifested cardiovascular insults in patients with known cardiovascular disease given electroconvulsive therapy. Kaldech et al. reported treating 50 patients with cardiovascular disease, including one case of definite

coronary artery disease, with no untoward effects (12) Bankhead et al. successfully treated 4 patients with old coronary thrombosis and 3 with histories of previous congestive failure (4). Shields et al. analyzed results of electroconvulsive therapy in 89 patients with cardiovascular disease, including 3 with previous coronary occlusions (having electrocardiographic evidence). They report 2 cardiovascular deaths, one due to coronary occlusion and one due to a cerebrovascular accident. They concluded electroconvulsive therapy could be given in all of these patients except those with coronary artery disease, but even here there was no absolute contraindication (20). In our series 3 patients with hemiparesis (old cardiovascular accidents) have been treated. In 2 of these, hemoptysis developed during the course of treatment. Thorough investigations did not reveal the cause of the hemoptysis. In one patient who developed hemoptysis, further electroconvulsive therapy was not administered since there had been no improvement of the mental symptoms after four treatments. However, consultations and examination failed to reveal any significant connection between the electroconvulsive therapy and hemoptysis. Following bronchoscopic clearance electroconvulsive therapy was continued in the other patient having hemoptysis and he recovered after a total of five treatments and was discharged with no further complications.

Studies of the effect of electroconvulsive therapy on cardiovascular physiology would also lead one to expect these results. The convulsion begins with a maximum forced expiration with elevation of the diaphragm and narrowing of the intercostal spaces. The resulting increased intrathoracic pressure acts to protect the heart in two ways: (a) any increased intracoronary artery pressure is equalized externally, tending to prevent capillary rupture; (b) blood from the periphery is prevented from returning to the great veins and right auricle (manifested by greatly increased peripheral venous pressure). As a result of the latter, the heart remains smaller than normal and its work is decreased during the convulsion itself. The convulsion ends with a maximum inspiration, and as respiration and normal intrathoracic pressure return, the blood, formerly locked up in the periphery, rushes into and distends the great veins and right auricle. It is in this stage, in the first minute after the convulsion, that the stress of electroconvulsive therapy is felt by the heart (1). The oxygen debt accumulated during the apnea of the convulsion also increases cardiac work at this time, as manifested by increased circulation time and cardiac output. However, as this latter phase requires many minutes, the cardiac work is spread out in time and "usually does not reach a dangerous level at any one time" (1). Electrocardiographic studies support these concepts.

During the convulsion, the electrocardiogram remains normal, but with cessation of the convulsion, a great variety of vagal arrhythmias are seen. The distention of the right auricle and great veins by the returning peripheral blood, stimulates vagal nerve endings, resulting in arrhythmia (2). To diminish the strain on the heart, Altschule recommends the use of curare. By lessening this intensity of the convulsion, the amount of blood locked in the periphery, and the oxygen debt accumulated, are both diminished (1). Curare preparations having impurities tend to increase the vagal effects mentioned above; therefore, Altschule prefers preparation free of impurities which are just as effective (3). Bankhead and co-workers, in addition to curarization, recommend atropinization (to prevent vagal arrhythmia) in all cases of organic heart disease, in all patients over 45 years of age, and in those patients having vagal reactions to electroconvulsive therapy (4). He also stresses selecting the correct shocking current initially, as several shocks result in a deeper cyanosis. Hejtmorgih stated that early post-convulsive oxygen is of value in preventing arrhythmia. He also recommends atropinization whenever the pre-shock electrocardiogram shows a prolonged P-R interval.

Diseases of the Respiratory System. In our series we encountered only 9 patients with chronic pulmonary disease—2 with pulmonary tuberculosis (both stable), 2 with pneumoconiosis (x-ray diagnosis), 2 with bronchial asthma, 2 with emphysema (senile), and 1 with bronchiectasis. These patients received from six to fifteen (maximum) electroconvulsive treatments and no complications were encountered.

In reviewing the literature, in this group of diseases, pulmonary tuberculosis appears to be the cause of most concern. The development of the spread of tuberculosis during electroconvulsive therapy has been reported, but only in a small percentage of the total number of patients with tuberculosis receiving electroconvulsive therapy (20, 26, 7). Williams and Barrera feel that tuberculosis is not a definite contraindication, and Close states that electroconvulsive treatments may be given in carefully selected cases where delay in electroconvulsive treatment would reduce the possibility of a good prognosis in the mental illness, or where there is the possibility of alleviating emotional symptoms which interfere with the state of the tuberculosis (7).

In this series there was only one patient with an acute respiratory disease. A 55-year-old, severely depressed female involutional psychotic was admitted to the hospital with a low grade fever. Physical examination was essentially negative except for blood pressure of 170/110. The fever was attributed to dehydration from failure to eat and drink for some time. She reacted poorly to electroconvulsive treatments, being

even more retarded and depressed, and after the second treatment her temperature rose slightly. That afternoon she became cyanotic, dyspneic, and went into peripheral vascular collapse. She responded poorly to fluids, oxygen and antibiotics and 24 hours later died. Autopsy revealed massive bilateral lower lobe pneumonia. Electroconvulsive treatments were accompanied by increased salivation and perhaps bronchial secretion. As the seizure ends, there is a maximum inspiration, with possible aspiration of infective material. The consensus of staff opinion was that the pneumonia was masked in terms of clinical findings and that the electroconvulsive treatment might or might not have been instrumental in extending it to a fulminating degree. Causative organisms were not identified. The smaller bronchioles may become blocked leading to atelectasis. If infectious material is aspirated, infection may develop in these areas (1). Because of such possibilities Kwalwasser et al. stress the importance of dental hygiene, and of discontinuing therapy with examination for cause at the first sign of fever or respiratory involvement (14). They, and also Kaldech, list acute infection as a definite contraindication to electroconvulsive treatment. Wilbur recommends atropinization to decrease salivation and the dangers of aspiration (25). The case in this series points up the importance of these suggestions. It also points up the importance of pre-electroconvulsive therapy chest x-rays—at least if any fever of undetermined etiology is present.

Diseases of the Osseous System. Fifteen patients (1 male and 14 females) presented diseases falling into this category. There were 4 patients with old (several years) vertebral fractures, 2 with vertebral column arthritis, 1 with severe kyphosis of the dorsal spine, 2 with generalized chronic carcinoma with metastases to ilium and acetabulum, 1 leg amputation for hemangiona with bone involvement, 1 childhood rickets with achondroplasia, and 1 hemilaminectomy (for ruptured disk)—two years old. These patients received from three to 31 electroconvulsive treatments with no related complications. The most unusual case was a 52-year-old depressed female who fell and fractured her hip while in the hospital. She refused all food and developed nutritional edema and ascites. Because of her rapidly progressive downhill course, a series of five electroconvulsive treatments with the routine curare premedication was given in an effort to bring her out of her depression and to improve her eating. There were no complications from the shock therapy, but her emotional status was unchanged. She was transferred to a general hospital, but she continued on her downhill course and, after several weeks, died. Autopsy revealed only findings of generalized cachexia.

Our findings support the claims of others (13, 18, 6, 17) that with adequate and proper care in therapy, electroconvulsive treatments may be given, though not without risk, even in the presence of many bone diseases.

Diseases of the Nervous System. Eighteen patients presented diseases considered under this heading. Two patients had old anterior poleomyelitis, 3 posterolateral column disease, 1 facial palsy, 2 multiple sclerosis, 2 parkinsonism, 1 Friedrich's ataxia, 1 postencephalitic psychosis, 1 marked confusion and amnesic state after previous electroconvulsive therapy, and 1 syncope after electroconvulsive therapy. One patient had an undiagnosed disease of the central nervous system, with cerebral atrophy shown in pneumoencephalographic studies. Three other patients do not actually belong in this review but are included because of their related interest—the development of spontaneous convulsions after electroconvulsive therapy in patients previously physically well. The patients in this group received from three to 20 electroconvulsive treatments with no ill effects other than the 3 who developed spontaneous convulsions. Spontaneous convulsions following electroconvulsive treatments, while rare, have been reported in the literature by many (19, 10, 26). Most reports are concerned with "late" convulsions, beginning weeks after shock therapy had ended. Pacella and Barrera (19) did electroencephalograms on 500 patients before electroconvulsive therapy was begun. Two of these patients developed spontaneous convulsions (six and one-half and eight weeks after shock therapy ended). Both showed abnormal electroencephalographic patterns before electroconvulsive therapy was begun and greater abnormalities afterward. Pacella on this basis felt that epilepsy was not produced by electroconvulsive therapy but was precipitated in individuals predisposed to it by other (undetermined) factors. However, many patients demonstrated abnormal electroencephalogram patterns before electroconvulsive therapy and did not develop spontaneous convulsions, and aged patients show abnormal electroencephalograms for about six weeks after electroconvulsive therapy. A completely satisfactory explanation for these spontaneous convulsions has still not been given.

Metabolic Disorders. Six patients with diabetes mellitus (3 males and 3 females) and 2 patients with thyrotoxicosis (both female) received six to 46 electroconvulsive treatments. No complications arose. In none of these patients was the disorder severe or out of control before or during electroconvulsive treatments.

Other Disease Entities Encountered. One case each of otosclerosis, detached retina, conical cornea, inguinal hernia, soft tissue injury (elbow), pregnancy (third month), increased bleeding time, polycy-

themia, and 2 cases of peptic ulcer are included in this study. One patient with microscopic hematuria received electro-convulsive treatments without any ill effects. Investigations were carried out, but a satisfactory cause for the hematuria could not be demonstrated. It was believed that the patient had chronic glomerulonephritis. These patients received six to 20 electroconvulsive treatments with no complications developing.

Deaths. During the seven year period covered by this study there were three deaths that might have been associated with electro-convulsive therapy. A 44-year-old female with compensated rheumatic heart disease developed a severe compression fracture of T_4 during the third electroconvulsive treatment. Since the compression was relatively high, the consulting surgeon advised traction and then a body cast. The patient seemed to accept the accident in spite of having made only a beginning improvement in her depressive state. She was hostile to her aunt and she was known to have suicidal preoccupations. There was no sensorium or neurologic defect but on the seventh day after the third electroconvulsive treatment and application of the cast she was found dead on the floor beside her bed. The consensus was that she had fallen in trying to get up. She had been seen 20 minutes earlier and was apparently in no distress then. Postmortem examination failed to reveal a definite immediate cause of death or other significant findings except the fracture, an acute dilatation of the heart, and cerebral and pulmonary congestion.

A second death occurred in a 19-year-old male catatonic markedly excited schizophrenic who went into circulatory collapse after four electroconvulsive treatments, did not respond well to vascular supportive therapy, and died one week later. Postmortem examination revealed a large infarct of the right lung and pulmonary congestion. There appeared to be no specific relationship between the physical findings of infarct as the cause of death (autopsy) and the electroconvulsive therapy.

The third death was the pneumonia case discussed previously. In none of these cases was there demonstrated any clear relationship per se between the electroconvulsive treatments and the actual deaths, except possibly in the case of the pneumonia.

Cases in Which Electroconvulsive Therapy Was Considered To Be Contraindicated. In this series, 18 patients who might otherwise have received it had they not had various physical defects were not given electroconvulsive therapy. Of these, 11 improved with other therapeutic methods which would be considered symptomatic, including the use of benzedrine or amphetamine and psychotherapy in the controlled hospital atmosphere. The remaining 4 were unimproved.

Of this group of 18 patients, 7 ranging in age from 53 to 84 pre-

sented findings of severe hypertensive disease, previous cerebrovascular accidents with residual findings, hypertensive retinitis, myocardial infarct, or severe cardiovascular changes including left axis deviation. One patient was not given electroconvulsive therapy because of a detached retina, another because of persistent auricular fibrillation, another because of extensive, old vertebral fractures, and another because of malignant metastases to the spine. One patient, aged 37, had a Potts fracture which was considered to be a contraindication. In this case electroconvulsive therapy was not urgently needed and so was postponed until a later date. Another patient, aged 84, had a moderately severe posterolateral sclerosis and another patient had an extensive spinal arthritis together with hypertension.

It is now believed that certain of these patients might have been given electroconvulsive therapy without complications, but at the time it was decided that their physical defects were sufficiently severe to be considered as contraindications.

Three other patients were considered to be too incapacitated for treatment and this opinion we continue to hold. One female, aged 74, with complete hemiparesis and generalized debility, one male, aged 62, with arteriosclerotic cardiovascular disease with decompensation and arrhythmia, and one female, aged 38, with parkinsonism, rheumatic heart disease, diabetes mellitus, arthritis with fracture of right femur.

Summary and Conclusions

1. In the seven year period from February 1946 through January 1953, 124 patients with physical disease constituting possible contraindications to electroconvulsive therapy were nevertheless given this treatment, after careful deliberation because of the severity of mental illness.

2. For this study the cases were subdivided according to the presenting somatic disease, and each group analyzed individually. Serious complications, possibly related to the electroconvulsive therapy, developed in only 2 of the 124 patients. One patient with arteriosclerotic heart disease developed a myocardial infarction and one patient died from lobar pneumonia. These results indicate that although electroconvulsive therapy is usually well tolerated, even by patients with severe physical disabilities, it cannot be used without appropriate caution.

3. The results of this study are compared with results of similar studies reported in the literature, and are correlated with what is known about physiologic changes occurring during electroconvulsive therapy.

4. The low incidence of complications in these patients is in agree-

ment with the results of others and supports the growing opinion that there are few absolute physical contraindications to electroconvulsive therapy, and that the risks of continued mental illness in the presence of physical illness must be balanced against the risks of electroconvulsive therapy in each individual case.

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SCHIZOPHRENIA: A REGRESSIVE PROCESS OF ADAPTATION*

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We would like to draw attention to a similarity of pathophysiologic and psychopathologic phenomena occurring, on the one hand, during certain stages of the schizophrenic process, and, on the other, during certain stages of the general adaptation syndrome (G.A.S.) of Selye (31, 32).

For example, the sympathicotonic anxiety states of acute schizophrenics during early phases of their illness seem to be characterized by somatic features similar to those which are present in nonschizophrenic subjects who have been exposed to stress (27) and are in the shock-phase of the alarm reaction of the G.A.S. Further, the vagotonic contrashock phase of the alarm reaction of the G.A.S. (31, 32, 33, 34) shows again a number of symptoms (14) described as concomitants of the acute catatonic phase of schizophrenia, e.g.: decreased B.M.R. (31, 24); decreased body temperature (31, 24); raised protein catabolism (16, 17); raised uric acid excretion (5, 17); lowered hippuric acid excretion after ingestion of sodium benzoate (9); reversible fatty infiltration of the liver (26); raised blood-lactic acid (31, 20); lowered "reduced" blood glutathione (2, 6); shortened blood clotting time (4, 18); acidosis (37); hypochloremia (30); slightly reduced glucose tolerance (15, 5, 6); hypoadrenalism (28, 29); gonadal atrophy (20); vagotony (24); loss of weight (1); increased diuresis (20); elevated N.P.N. (16, 17), and decreased thyroid function (8, 23).

Finally psychic and somatic symptoms and behavior of chronic (burned out) schizophrenics seem to simulate in many respects the stage of resistance of the G.A.S.‡

In an oversimplified and generalized assumption, it might be stated that certain phases of the G.A.S. seem to be permanently present during the schizophrenic process, caused mainly by a *genetically inherited* (7, 21) abnormal low threshold toward stress.

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‡ The rare cases of lethal catatonia would fit in this scheme if we assume the quick transition of these patients from the contrashock to the exhaustion phase of the G.A.S.

Speculating along these lines, it might be that minimal amounts of toxic substances, supposed to be produced at the beginning of the alarm reaction during early stages of the schizophrenic process, are also involved in the process of maintaining the G.A.S. as well as to release and maintain the well known psychopathologic disturbances. Model experiments on normals with certain psychotica and eidetica, such as mescaline (3, 13) or lysergic acid diethylamid (LSD) (35, 12), support the above hypothesis with some indirect evidence. It was also possible to demonstrate the presence of a toxic factor present in the blood and urine of schizophrenics (11). The serum and urine of acute schizophrenics was tested, in follow-up studies, on larvae of *Xenopus levis Daudin* bred at 20° C; 10-20 ml/liter of serum or 25-50 ml/liter of urine, if added to the water milieu of 14-day-old larvae, caused a significantly higher mortality than the serum and urine of healthy controls in the same dilutions. Longitudinal studies revealed a parallelism between psychic status of the patients and toxicity of their serum and urine, respectively. Spontaneous remission or successful treatments gradually diminished the toxicity to near normal values.

The toxicity of serum of schizophrenics disappears after exposure to 56° C for 30 minutes, or after dialysis, whereas urine maintains its toxicity even after being boiled for 30 minutes.

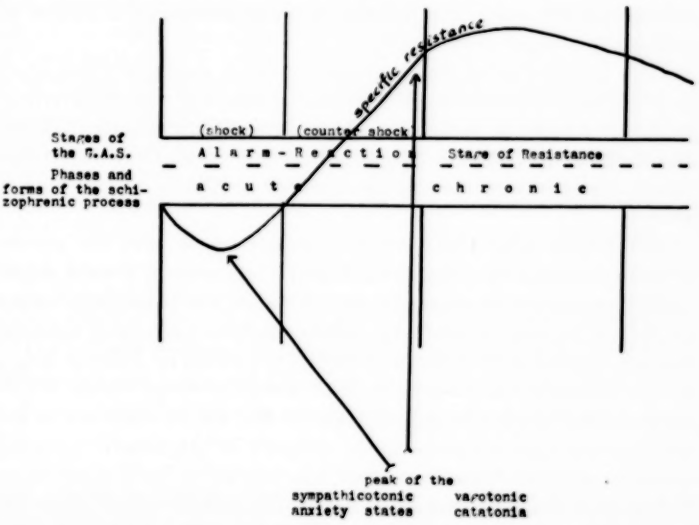

FIG. 1.

At present, another possibility cannot be excluded, namely that the higher toxicity of schizophrenic serum and urine might be a concomitant of the G.A.S. as such. This type of speculation might also be supported by experiments showing an increased toxicity on tadpoles of the blood of normal women with hyperemesis gravidarum in their early stages of pregnancy or of normal women in their last months of pregnancy (10). Both of these conditions are also stress situations but apparently less strong than that represented by the acute catatonic phase. Consequently, the toxic action of the blood, displayed during hyperemesis or late pregnancy, is definitely less pronounced (10) than is the toxicity of blood from acute schizophrenics in the catatonic phase (11). The hyperemesis patients display a slight increase in toxicity if compared with healthy controls, whereas the toxicity indices of females in their last months of pregnancy lie between the indices of nonpregnant normals and acute schizophrenics. The serum of patients with cirrhosis or carcinoma of the liver shows also similar medium-values.

Thus, the toxicity to tadpoles of serum seems to be positively correlated with the degree of stress.

Comment

Since, as a result of many years of schizophrenia, the patient's threshold toward stress is significantly raised, schizophrenia might be defined as a regressive process (14) of adaptation towards philogenetically more archaic and less vulnerable patterns of organization in the psychic as well as the biologic sphere.

As a consequence of the above assumption, it might be stated, that:

(A) Early stages of the schizophrenic process might be characterized by the permanent or periodic presence of the G.A.S. as a result of the inherited low threshold of the patient toward stress. Thus the patient during approximately the first two years of his illness is under undue stress.

(B) As the schizophrenic process progresses in time, the patient develops a resistance (tolerance), higher than normal, toward stress.

With reference to statement (A), some of the stress experiments carried out on schizophrenics by different authors (36), using different stressors applied with different strength on patients in different stages of the schizophrenic process, in some instances reveal results which are not reproducible (36, 25). It might be that the inconsistency of the results is not caused merely by the difficulty of quantitative norming of the experimental conditions, but also because in such experiments both groups, the schizophrenic as well as the normal control group (or neurotic), were stressed in the same way, not taking into account that

schizophrenics in their acute stages are already under stress before the experiment.

Studies by Hoffer (19) might also serve our purposes in adding evidence in support of our above-mentioned theory. He administered 3 mg. of atropine to more than 100 acute and 100 chronic schizophrenics; 80 normals and neurotics served as a control group. As was to be expected, 95 per cent of the latter group experienced the 3 mg. of atropine as a stress and reacted with the sympathicotonic shock phase of the alarm reaction of the G.A.S. (elevation of the systolic blood pressure). Ninety per cent of the acute and 80 per cent of the chronic schizophrenics, however, as well as another smaller group of neurotics already under an additional stress, all being either in the contrashock phase or in the phase of resistance of the G.A.S., did not experience the 3 mg. of atropine as a (real) stress and revealed only a slight increase of their more or less vagotonic stage (decrease of the systolic blood pressure). With smaller groups of acute schizophrenics and other diagnostic groups (neurotics, psychopaths, depressions), it was found that after atropine there was no change whatever in total or differential count in schizophrenia. With the other groups the eosinophil count after two hours decreased 40 per cent, the lymphocyte count 20 per cent, the polymorphonuclear count increased about 10 per cent, and the rhabdocytes dropped markedly. The total white count decreased slightly.

With reference to statement (B), let us illustrate the abnormally high resistance of chronic schizophrenics toward stress—developed during and because of the schizophrenic process itself—by the experiments of Lucy (22).

He found that more than the lethal dose of histamine (10 mg.) was supported by these patients without any typical stress reaction. The "resistance" (tolerance) of the chronic schizophrenics toward histamine, a general stressor, seems to be roughly proportional to the duration of the schizophrenic process.

Summary

Certain stages of the schizophrenic process seem to correspond with certain stages of the general adaptation syndrome.

The initially abnormal low threshold toward stress is raised during the schizophrenic process to an abnormally high one.

A toxic factor for tadpoles was shown to be present in the serum and urine of acute schizophrenics.

Whether the inherited low threshold toward stress might play the main role in maintaining the schizophrenic process, or whether there might be certain toxic substance(s) involved in the mediation of the

schizophrenic process, i.e., the "regressive adaptation syndrome," cannot yet be decided conclusively.

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A CONTRIBUTION TO THE PSYCHOBIOLOGIC APPROACH IN SCHIZOPHRENIA*

A Report on Two Cases of Schizophrenia with Pseudo-eunuchoidism

OSKAR GUTTMANN, M.D.

Psychobiology should be considered as a special branch of biology attempting to study systematically all significant phenomena of the human psyche. The logical approach in psychobiology would be an integrated study of the individual as a whole because of the twin-like body-mind relationship rather than a separate consideration of the physical or psychic facets of the personality.

The problem presented is one of providing an analysis of the intangible phenomena of the psyche. Modern psychobiology attempts to approach the matter by correlating the psychologic occurrences with the physiologic events. This approach goes beyond the original working principle of Adolf Meyer, which consisted of explaining human behavior in terms of adaptation.

Recent developments in neurophysiology and endocrinology have greatly increased the number of avenues available for exploring this subject. Modern psychobiologic research in schizophrenia is still in an early stage. There are relatively few serious studies. Among them, the most important are:

1. Those of Angyal about the general physiologic hyporeactivity in schizophrenic withdrawal.
2. The studies of the deficient oxygen metabolism in schizophrenia.
3. The research work of Sayers, Pincus, Hoagland, Hoskins, and Malamud regarding the function of the pituitary-adrenal system in psychosis.

A problem of paramount importance in this type of research is the relation between the gonadal deficiency and schizophrenia. One of the oldest organogenic theories of dementia praecox, expressed by Kraepelin in 1888, is that a deficiency of the gonads is an etiologic factor in this illness. Dercum saw a connection between early defective development with hypofunctioning of all endocrine glands. Fraenkel observed

* Presented at the Upstate Interhospital Conference of the Department of Mental Hygiene, Syracuse, The Psychopathic Hospital, Apr. 7, 1953.

infantile genitalia in 72 per cent of his cases of dementia praecox. Sippel reported hypoplasia and hypofunction of the gonads in a high percentage of cases. His efforts to treat his female patients with ovarian transplants from animals were unsuccessful. Confirmatory observations and results were reported by Poetzl and Wagner. Muenzer described a case of a young schizophrenic with delusions about his genitalia, who committed suicide. The pathologic report revealed enlargement of the thymus, spleen, lymph nodes, hypoplasic adrenals, atrophy and degeneration of the testes, reduction in number of the pancreatic islet cells, and eosinophilic adenoma of the hypophysis. McCartney had an opportunity to study 158 male schizophrenics and found almost 60 per cent with genital hypoplasia.

This author expressed the opinion that schizophrenia is primarily an endocrinopathy due to hereditarily defective somatoplasm. In schizophrenia, fully developed endocrinopathies are not generally observed. Hoskins and Sleeper found evidence of endocrine disturbances in 50 per cent of their schizophrenics and almost the same percentage of improvement following hormonal treatment. The best results were obtained in the catatonic phase, and the poorest results in the paranoid phase of schizophrenia. The authors believe that this difference in results may be related to the high incidence of thyroid deficiency in the catatonic phase and of pituitary deficiency in the paranoid phase. Hoskins has not offered any opinion as to whether the obviously disturbed psychosexual development of the schizophrenics is the cause of this illness.

In the cases of Nolan Lewis, 16 out of 19 male patients had grossly soft and undersized testes with a complete absence of spermatozoa when biopsy specimens were examined microscopically. Matsumato reported regressive atrophy in the testes of schizophrenics and formulated the theory of biochemical changes in the nucleus of germ cells of the neurons. He stated that an inborn defect in the germ-cell nucleus may lead to a disturbed functioning of the neurons. Mott believed that the testicular atrophy occurring in schizophrenic adolescents is a protective mechanism of nature designed to prevent the reproduction of a degenerate progeny. The testes of schizophrenics probably do not grow much beyond the pubertal period but there is probably enough interstitial tissue which survives for the development of the secondary sexual characteristics. This is not physiologically impossible, because Pezard observed experimentally in the fowl that 1/30th of the testicular tissue would supply a sufficient quantity of hormones for the development of secondary sexual characteristics. Hemphill and Reiss studied this problem by performing biopsies of the testes in cases of schizo-

phrenia and found that a high percentage of testicular atrophy occurred which varied with the severity of the psychosis. The atrophy was rarely found in the paranoid phase of the disease and could not be correlated with the age or with the urinary 17-ketosteroid excretion.

Two cases of schizophrenia will be presented from a psychobiologic standpoint:

Case Reports

Case 1. L.R. is an 18-year-old white male. The psychiatric diagnosis was schizophrenia, catatonic phase. His mother died when he was an infant. His father is a severe alcoholic and a tyrannical hypochondriac who was often abusive toward his family. Seven other siblings of the patient showed neurotic features emphasized by emotional instability and conflicts over family problems. The boy was raised by a housekeeper who neglected the children and mistreated them. It is alleged that she often put soap powder and garbage in their food. She developed a psychosis and was admitted to a State hospital. L.R. was placed in one foster home after another, including the homes of some of his married sisters. He was never treated with affection and was forced to do hard work in the household. He was merely fed and clothed and found little enjoyment in life. At a later date a younger married sister took compassion on him and brought him into her home. The boy had developed a hostile attitude because of his previous rejections and got along indifferently at first in this warmer environment. He was reticient and avoided people, especially girls. The patient enjoyed drawing and liked to work on farms during the summer months. His younger sister was successful in readjusting him socially. He became a member of the Young People's Group in the Methodist Church. He was in the ninth grade of school at that time and was considered an excellent student. He also joined the school's baseball team.

In February 1951, L.R. complained of malaise and pains around his eyes. A physician advised rest from school, a tonic and sedation. The patient was cared for at home by his sister. He improved enough to be able to return to school the following year.

In March, 1952 during an English class the patient became disoriented and began to talk about baseball; then ran to the wall and banged his head against it. The physician gave him sedation. At home he was more quiet, but laughed in an idiotic manner and stared into space most of the time. He became restless, confused, and incoherent and was brought to the hospital in an excited state. On admission and during the next five weeks the patient presented the same mental picture of confusion and excitation with great overproductivity, auditory and visual hallucinations of God, the Blessed Virgin, and the Devil. He expressed bizarre ideas about Captain Marvel, Ken Murray, Uncle Joe, and Uncle Sam.

In March and April the patient was given a total of 30 electrocoma therapy treatments, 29 of which resulted in grand mal seizures. The pa-

tient improved slightly and became more manageable and no longer needed restraint.

FIG. 1.—Case 1. (Before and after treatment with Oreton)

In June he received 16 injections of insulin or a total of 2206 units which resulted in 50.7 hours of hypoglycemic shock. He did not react well to the insulin treatment and by the end of July he had received 12 more electrocoma therapy treatments. He improved markedly and recovered to such an extent that his release was considered. Shortly thereafter he suffered a relapse and became excited again, requiring restraint and electrocoma therapy. He developed delusions that he was a very bad man and identified himself with heroes of various comic strips. After improving somewhat, the patient was submitted to biologic studies. A physical examination revealed the following: height, 5′4″; weight, 105 lbs.; type, leptosomatic, and voice, normal for his age. He had no gross deformaties but had a slight degree of prognathism, flat feet, and a slight disproportion between the length of his arms to the rest of his body. His skin was white and smooth. His appearance was somewhat effeminate. The hair on his head was thick but was absent on the face, chest, abdomen, pubic region, and extremities. He had never shaved. There was little hair in the axillary pits,

slight hair growth around the base of the penis, and no hair at all on the scrotum. A slight pigmentation line from the umbilicus to the symphysis pubis was noticed. The length of the penis approximated that of a 15-year-old boy. The testicles were small, just slightly larger than almonds.

A glucose tolerance test was performed with 100 Gm. given orally. The results were: fasting—120; one-half hour—147; one hour—151; two hours—136; three hours—114, and four hours, 120 mg. per cent.

In evaluating the function of the pituitary-adrenal system the epinephrine test was performed three times using a 1:1000 epinephrine solution. Differential white blood counts and direct eosinophil determinations were done at one- to three-hour intervals. All curves were within normal limits. In one determination the direct eosinophil count dropped from 200 per cubic millimeter to 138 after one hour and to 145 after three hours. In another test a similar decrease from 148 to 46 per cubic millimeter during the same time interval was observed. The quantitative determination of the 17-ketosteroids in the 24-hour urine were 19 mg. and following the epinephrine test there was a reduction to 12.4 mg. A functional test of the vegetative nervous system activity was performed using mecholyl, a cholinergenic substance, but violent vomiting caused the test to be interrupted and was later performed with 1 cc. of adrenalin 1:1000 intravenously. Blood pressure and pulse rate determinations were done every two minutes for the first 15 minutes following the injection and every five minutes during the next half hour. Electrocardiograms were done before the test four minutes following the injection and at the end of the test. The interpretations were all within normal limits and were almost unchanged. There was very slight alteration in the pulse rates. The blood pressure increased by only 10 mg. of mercury. Cholesterol was 228 mg. per cent; basal metabolic rate was +10; electroencephalograms abnormal, and the recording showed a diffuse cortical dysrhythmia consistent with a convulsive disorder of the grand mal type. The psychologic tests given were: Wechsler-Bellevue Intelligence Scale, Rohrschach test, Goldstein-Scherer object and color form sorting test, Bender visual motor gestalt test, and a make-a-picture-story test. The following were the findings: full-scale I.Q., 90; low average ability; rigid affect; emotionally labile due to anxiety in the sphere of sexual adjustment; defective judgment with failure to take into account important details in his environment, and a schizoid personality.

In October and November of 1952 the patient received 12.5 mg. of Oreton*, twice weekly during the first month and 25 mg., twice weekly during the second month. The patient showed marked improvement following hormonal therapy. At the institution where this treatment was administered the patient was given an honor card and was allowed to work in the warehouse of the hospital. It was felt that the hormonal therapy was a contributing factor in making the patient more vigorous and mature. He was more alert and spontaneous and showed a greater degree of stability in his behavior. He had a more co-operative attitude and coherent normal

* Testosterone Propionate, Schering Corporation.

ideation without delusions and hallucinations. He made rather sensible and serious remarks about girls, admitting his penile erections and sexual cravings. The patient now seemed to be ready for release from the hospital.

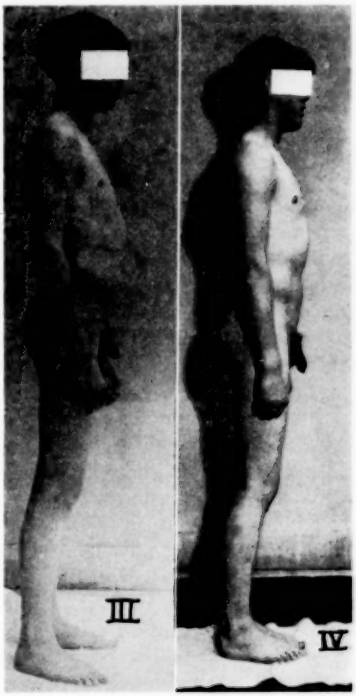

FIG. 2.—Case 1.

The last physical examination was given on Dec. 2, 1952 and revealed a more mature young man and one who had lost many of his effeminate features. His musculature and skin became more masculine in type. He now had hair on his legs, forearms, in the pubic and scrotal regions, and there was the appearance of fuzz on the upper lip. The hair on his head had become darker in color and there was a change in the texture. His weight had remained nearly the same although there had been a noticeable change in its redistribution. The abdominal fat had been lessened and there was a more marked accentuation of the pelvic girdle. There was an increase in the length and diameter of the penis and the testicles had increased in size and were normal for his age.

In this case the prognosis was favorable.

Case 2. B.S. is an 18-year-old white male. The psychiatric diagnosis was schizophrenia, hebephrenic phase. The patient's parents and 3 brothers

were in good health. A sister, 21 years old, had been in Binghamton State Hospital for two years, from 1948 to 1950, with a diagnosis of schizophrenia, hebephrenic phase.

B.S. walked at the age of 14 months and talked at the age of three years. When he was five years old another brother, R., was born. The patient has always been jealous of R., who is more sociable, more efficient in everything, and plays chess, which the patient refused to learn from his father. He attended school only as far as the sixth grade because he was backward in his studies. He liked a 12-year-old school girl but hardly talked to her. After having quit school he worked in a bowling alley, but was fired after a short time. He was very childish and never asked any questions, had no interest in sports, music, or in dancing. He did not smoke or drink, was seclusive, shy, and was a bad mixer. He liked to walk along the roads to watch building construction. He worked with a chemistry set in the cellar, read many history books and mystery stories. When he met other schoolboys, with whom there was poor rapport, he would ask them questions concerning historical events about which he had probably read something recently. When the boys did not know the answers, he became angry.

His mental symptoms first began to appear about two years prior to his hospitalization. His emotional outbursts became more frequent. He began to feel that he was disliked and unwanted and became completely disinterested in his surroundings. He began to be unclean and even refused to take a bath. In his seclusiveness he began to hear voices calling him bad names and thought that monsters were after him. He began to react violently to his delusional and hallucinatory experiences. On one occasion he threw a glass of milk at an imaginary monster. On another occasion he broke a lamp to defend himself against an imaginary animal. His sleep was disturbed by nightmares. He would wake up at night screaming and laughing. He became forgetful. When his destructive tendencies assumed a dangerous character, he had to be hospitalized.

In the hospital he was confused, resistive, unco-operative, overactive, idiotic, manneristic with an inappropriate and shallow affect, echolalia, echopraxia, and stammered slightly. The patient received 20 electrocoma therapy treatments between April and June 1952. His response was not entirely satisfactory although he became more quiet, more co-operative, and less manneristic. He lost his confusion and hallucinations, echolalia, echopraxia, and was only vaguely delusional. When sex was discussed he was embarrassed and giggled. He remained silly and autistic. His stammering increased. He accomplished nothing in the occupational therapy class. He spent his time reading comic books. On admission the physical findings were as follows: height, 5'6"; weight, 98 lbs.; type, leptosomatic. His skin was tender and smooth and he appeared effeminate. He had round shoulders with prominent scapular angles and general underdevelopment of the skeletal musculature. There was questionable congenital subluxation of the left hip joint. His arms were slightly longer than normal.

He had fine-textured hair on the scalp. Hair was absent on his face, chest, and abdomen. He had never shaved. He had little axillary hair but had a normal amount in the pubic and scrotal region. The penis and testicles were almost normal for his age. The patient stammered, had a weak grip, and clamminess of the hands and feet. The voice was of a childish pitch and his vision was myopic. His finer movements lacked deftness.

Fig. 3.—Case 2.

The glucose tolerance test was performed with 100 Gm. of glucose with the following results: fasting—90; one-half hour—154; one hour—102; two hours—92; three hours—90, and four hours—90 mg. per cent. The epinephrine test was performed three times and the same abnormal response in the direct eosinophil count was found. One count was particularly interesting as it showed a remarkable increase of eosinophils from 100 per cubic millimeter before the epinephrine injection to 300 afterward, instead of the normal drop of more than 50 per cent. The quantitative determination of 17-ketosteroids in the 24-hour urine specimen was 9.8 mg. and following the epinephine test was 5.96 mg. Cholesterol was 200 mg. per cent and the basal metabolic rate +9. The functional test for the vegetative nervous

activity was performed by administering 0.8 cc. of adrenalin 1:1000 intravenously followed by three electrocardiograms, six blood pressure rates, and six pulse tracings. The electrocardiograms were normal and did not show any change. The blood pressure did not change at all immediately following the injection of adrenalin, but approximately six minutes later there was a slight drop from 114/74 to 100/60, values which were maintained beyond a half-hour following the injection. The pulse rate rather than increase dropped from 100 to 84. Electroencephalograms revealed abnormal recordings which is consistent with "brain trauma." The same psychologic tests were administered as in the first case; in addition, the TAT was given by the writer. This showed marked silliness and immaturity of the patient. A full-scale I.Q. of 63 was recorded and the psychologist felt that he was a moron. During the tests he showed an inability to think of concepts on an abstract level; frequent stammering, trembling, and idiotic laughing were noted. He had no insight and did not seem to be concerned about his fate. He made a fairly good adjustment in the protected environment of the hospital.

In October and November the patient received the same treatment with

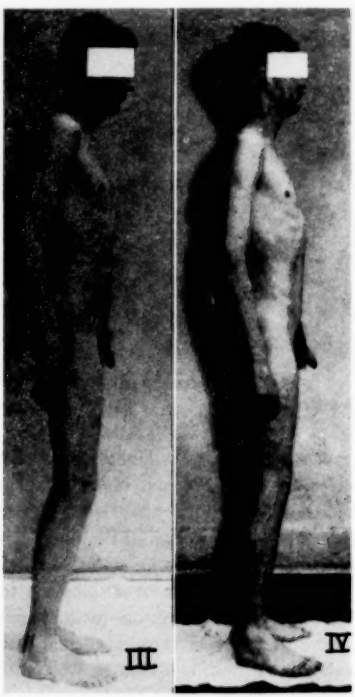

FIG. 4.—Case 2.

Oreton as the first patient. This patient did not show any essential changes. Mentally he was a little more pliable to discussions, less impulsive, less excitable and seclusive, more quiet, and more interested in doing things. However, he remained the same childish immature individual. The physical examination after treatment showed a slight increase in the size of the penis and testicles, an increase in pubic and scrotal hair, a remarkable hair growth on the legs and arms, but there was still no hair on the chest and abdomen. He had a beginning beard growth. The hair on the scalp became thicker and darker. There was no change in his voice.

In this case the prognosis was poor.

Discussion

In both schizophrenics we were obviously dealing with an interesting organogenic disturbance within the hypothalamic-hypophysial-testicular axis. Both patients were effeminate or androgynoid in character and had a leptosomatic constitutional type. They were physically underdeveloped, showed a failure of normal pubertal maturation, and were emotionally and mentally immature. B.S. is, in addition, a stammerer and became easily excited and angry when he encountered difficulty in expressing himself. Both present an unfavorable hereditary background with severe environmental stresses in L.R.'s case, and a long-lasting sibling rivalry in B.S.'s. Both felt rejected and had severe maladjustments and inferiority complexes. They had strong ungratified cravings for affection, hostility toward the world, and vague perceptions of failure. A nearly identical pattern of prepsychotic schizoid personality and antisocial behavior was found in both boys. Both displayed the schizophrenic reaction under similar conditions; namely, when they could no longer cope with the stresses of reality they sought refuge in the fanciful world of the child. B.S. and L.R. showed similar pictures in their psychotic episodes with hallucinations of persecution or grandeur, confusion or excitement. They also showed the same vicissitudes and aberations of behavior. The physical findings were also analogous in both cases. The skeletal changes in L.R.'s case were mild prognathism and flat feet; while in B.S.'s case there was a questionable congenital subluxation of the left hip joint (the x-ray was negative). In both cases the arm length was slightly greater than normal (by 3 cm.). Both patients were without hair on the face and showed a scanty growth on the chest, abdomen, and in the axillae. Variable hair growth was observed in the pubic and scrotal regions. The external genitalia were less developed in L.R.'s case and almost normal in B.S.'s case. Both patients displayed a lack of secondary male sexual characteristics.

Laboratory findings were also similar. Some values were within normal limits as might be expected and some were abnormal. The well-

known epinephrine test with direct eosinophil count was normal in L.R.'s case, but showed a repeatedly abnormal pattern in B.S.'s case. Here, instead of a normal eosinopenia of more than 50 per cent, we found a considerable eosinophilia. The 17-ketosteroid excretion of 24-hour urine was low in both patients in comparison with normal excretion of 20 to 50 mg. in the same age group. A distorted response pattern was also found. Instead of finding a quantitative increase of these substances following the epinephrine test, there was actually a decrease. We may assume that the pituitary-adrenal system does not function adequately in both patients. Also it is important that we make mention of the fact that recently more and more authorities question the diagnostic value of the eosinopenia in the epinephrine or ACTH test in the evaluation of the function of the pituitary-adrenal system. The same applies also to the quantitative determination of the 17-ketosteroids which, according to some authors, has no diagnostic significance. Hoagland and Pincus recommend instead the quantitative determination of the B-ketosteroid which is the precursor of the 17-ketosteroids and was found in increased quantities in the schizophrenics. The tests of the vegetative nervous system function with adrenalin showed a normal pattern in activity in L.R.'s case and a pattern of hyporeactivity in B.S.'s case. In both boys there were abnormal electroencephalographic tracings. The tracings in L.R.'s case suggested a convulsive disorder of the grand mal type; whereas, those in B.S.'s case were suggestive of "brain trauma." Clinically and anamnestically we could rule out such processes. This demonstrates once more the well known high frequency of abnormal electroencephalograms in schizophrenics (about 30 to 40 per cent).

Kennard et al. found abnormal electroencephalograms in 60 per cent of the schizophrenics and mostly in young patients with an early onset of psychosis, long duration, malignant clinical course, and a positive family history. The psychologic tests were of great help in understanding the personalities, their emotional reactions, and their mental capacities. The response to treatment was different in the 2 cases. L.R. had a better prognosis; however, both improved following electrocoma therapy. During hormonal therapy the beginning of a clear and rapid physical maturation accompanied by a more vigorous and alert function were noticed, especially in L.R.'s case.

Both cases may be considered in the pseudo-eunuchoid group; that is, individuals with normal or slightly subnormal external genitalia presenting underdeveloped secondary male sexual characteristics. This disturbance is considered as an end-organ defect due to a functional or organic failure of the androgen-forming structures. The American

Indian and the Mongols are racially determined pseudo-eunuchoids. These men cannot be termed hypogonads since this would imply a totally impaired testicular functioning with failure in the primary formation of spermatozoa. It is a well established fact that the functioning of the testes is controlled by the hypothalamus and regulated by two gonadotropic hormones of the anterior lobe of the hypophysis. One of these the FSH or follicle-stimulating hormone originating in the delta cells stimulates the maturation of the germinal epithelium up to the formation of spermatozoa and is aided by the secretion of the sertoli cells. The other the ICSH or interstitial cell-stimulating hormone which comes from the alpha cells stimulates the function of the Leydig cells of the interstitial tissues of the testicle that secretes the androgen substance responsible for the determination of the secondary male sexual characteristics. The ACTH originates in the beta cells and stimulates the adrenal cortex to secrete the cortins. Almost one-third of these comprise the 17-ketosteroids. The Leydig cells also secrete estrogens which inhibits the FSH and ICSH and probably stimulate the formation of the ACTH. The estrogen also has some relationship to the formation and maintenance of the skeletal tissues intervening mostly at the epiphysial-diaphysial junctions of the long bones.

Conclusions

In both cases one may suppose that there is an organogenic and functional failure in androgen formation by the Leydig cells. Biopsies have not been able to establish whether this failure is caused by primary defect resulting from a lack of ICSH or is a secondary process resulting from insufficient functional response or structural change in the Leydig cells. There exists also the possibility of an increased estrogen formation which inhibits the gonadotropic hormones and the adrenal cortex. This improvement was supported and enhanced by the substitutive therapy with testosterone. The question of how the mental improvement took place is impossible to determine at the present time. We assume today that testosterone, among other effects, increases the oxygen metabolism of the brain tissue. The relationship of this to psychic processes and to schizophrenia is, so far, beyond our present knowledge. A fascinating view would be to see our 2 patients in the light of the first holistic concept of Nolan Lewis. Their body type, physical and physiologic findings are suggestive of a cardiovascular hypoplasia and adrenal-cortical insufficiency. In 1923 Nolan Lewis suggested that the hypoplasia of the cardiovascular system and adrenal-cortical insufficiency have some connection with the regression and the inability of schizophrenics to cope with environmental stresses. The fact that schizophrenics have an

abnormal psychosexual development showing a fixation at or a regression to the narcissistic-homosexual level does not allow us to conclude that they have a disturbed ratio of estrogen and androgen titers or that these hormonal disturbances are necessarily etiologic factors in schizophrenia. They merely may display only one of the many effects of the unknown schizophrenic process upon one of the most vital systems of the organism—the endocrine glands. They also show us one of the distorted and hyporeactive physiologic processes of the schizophrenics. The recent eucorticism theory of Sayers and Sayers (1950) suggests that the metabolic turnover of steroids in the somatic cells may vary widely in the organism and effect differential physiologic responses that may be interpreted as abnormal under conditions of apparently normal physiologic steroid titers.

Thus the findings of this report, while they do not show any absolute correlation between titer of steroids and clinical picture, may illustrate the statement of Sayers. Thus we may see normal steroid titer accompanied by subnormal or even abnormal peripheral somatic response.

Summary

A brief summary of the literature suggests a relationship which may exist between gonadal deficiency and schizophrenia. Two cases of hypogonadal male schizophrenics, both 18 years old, are presented; one classified as a catatonic and the other classified as a hebephrenic. The histories of a breakdown and the clinical pictures are similar. The catatonic patient received electrocoma therapy and insulin shock, while the hebephrenic received only electrocoma therapy. After moderate improvement both patients were subjected to psychobiologic studies which included the following:

1. Physical examination with evaluation of the constitutional features, such as skeletal characteristics, hair growth, and genital development
2. Glucose tolerance tests
3. Tests of vegetative nervous function, using adrenalin
4. Serum cholesterol and BMR
5. Circulating eosinophil response to epinephrine (for evaluation of the pituitary-adrenal system)
6. Quantitative determination of the 17-ketosteroids before and after the epinephrine test
7. Psychologic tests
8. Photographs demonstrating the somatic features before and after treatment

The patients received treatment with Oreton for two months and

physical and mental improvement were noted, especially in the catatonic. In cases of schizophrenia in males associated with gonadal hypofunction manifested by a lack of secondary male sexual characteristics, the mental symptoms may also respond favorably to hormonal therapy.

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THE MENSTRUAL CYCLE AND INSULIN COMA TREATMENT IN SCHIZOPHRENIA*

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Mental illness may be characterized not only by distortion of psychosexual development but also by somatic manifestations of disturbed sex function. Thus menstruation, the topic of this study, was absent in 38 per cent of a representative sample of mental patients selected for electric shock treatment (1). Electric shock treatment itself may further modify menstruation if it has not been already suppressed by the illness (1, 2). Even though the menstrual disturbance might be only a secondary manifestation of disturbed central organization associated with mental illness (3), it was considered worth while to study the manner in which menstruation is modified by insulin coma.

Methods

The records of female patients who were treated by insulin coma at the New York State Psychiatric Institute between the years 1942 and 1952 were reviewed. Only patients who menstruated in the hospital preceding insulin coma were included in the study. Patients who in addition received other forms of treatment such as electroshock therapy were not included. Ninety-eight records of patients aged 13 to 49 were found to contain sufficient data for derivation of the facts necessary for the survey. Ninety-five of these patients were classified in the category of schizophrenia and these form the basis for the statistical analyses in this report. The diagnostic classifications and the conditions of improvement from treatment were derived from the final discharge notes even though the discharge of the patient from the hospital might have occurred several months following completion of insulin coma.

In this manner, any bias on the part of the investigators as to the effect of insulin coma was avoided. The procedure of treatment followed a standardized pattern in most patients. After a test dose for sensitivity to insulin, the dosage was rapidly increased on subsequent days of treatment usually leading to coma within the first week of

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treatment. The patient was then maintained on the minimum dose of insulin which would lead to coma. Coma was maintained for 30 minutes and then terminated by gavage or intravenous injection of glucose solution. Treatments were given six days a week and the patient was treated continuously until 50 comas had been produced. For various reasons and complications, 20 of this group of patients had less than 50 comas, and of these, 8 patients had less than 30 comas. The average number of treatments including the preliminary light coma and subcoma treatments was 50.8 treatments in the group of 95 schizophrenic patients.

Results

As anticipated from clinical experience, the disturbance in menstruation during insulin coma treatment was chiefly characterized by amenorrhea. Of the total of 95 schizophrenic patients, 61 had been in the hospital a sufficient length of time preceding insulin coma to have menstruated twice, thus providing a control cycle for comparison with cycles following the introduction of insulin coma. The average length of the cycles preceding insulin coma was 32.7 days with a standard error $= \pm 2.02$. The average cycle during insulin coma which was derived by dividing the number of days intervening from the last menstruation before insulin coma to the first menstruation following insulin coma by the number of cycles during this period was 42.6 days (S.E. $= \pm 3.02$) in the identical group of 61 patients. The difference between these means was statistically significant at the .01 probability level ($t = 3.01$).

Lengthening of the menstrual cycle was not necessarily an immediate reaction to insulin coma. In 54 patients intervention of insulin coma on the menstrual cycle failed to produce amenorrhea in the first cycle during treatment. The first cycle was 30 days or less in length, and in some patients even shorter than normal. In these patients the first cycle ran its course with little alteration and amenorrhea did not occur until the coma treatment continued into the second cycle. As a consequence of the diversity of reaction to coma, the average length of the cycle during which insulin coma was introduced (38.0 days, S.E. $= 3.50$) did not differ significantly from the cycle preceding coma treatment. An initial average cycle with a subsequent long cycle of amenorrheic proportions was the prevalent pattern, but the reverse, that is early amenorrhea with a following average cycle was not exceptional. Of the total group of 98 patients, 13 remained amenorrheic throughout insulin coma. In 51 patients the first cycle was shorter than the second, in 30 patients the first cycle was longer than the second,

and in 4 patients both cycles were of equal length. As far as could be determined, these patterns of the menstrual cycle bore no relationship to diagnositc subgroup, amount of insulin used, length of treatment, or age of patient.

Less insulin was needed to induce coma in older patients. However, the correlation coefficient between age of patient and the average amount of insulin used per treatment $r = -.21$, P.E. $= \pm .07$, was not statistically significant and might be at best considered as indicating a trend toward the use of less insulin to induce coma in older patients. The menstrual pattern as represented by the length of menstrual cycle was in no way related to the average amount of insulin needed for induction of coma.

Of far greater significance was the observation of a relationship between the length of menstrual cycle and the therapeutic effectiveness of the treatment. In the discharge notes, patients were classified into five groups of improvement. To achieve more valid statistical calculations and to avoid splitting of the limited number of patients into insignificantly small groups, the original five classifications were reduced to three grades of improvement: (a) recovered, which included the original recovered and much improved groups; (b) improved, which included the original improved and slightly improved; and (c) unimproved, which remained the same as the classification in the discharge notes. The first cycles of a group of 27 schizophrenic patients classified as recovered were significantly longer than the first cycles in the other two groups (see Table I). The difference between the average

TABLE I.—Improvement from Insulin Coma in Relation to Length of First Menstrual Cycle (A) and Average Menstrual Cycle (X) During Insulin Coma

	Number of Cases	*A*	*S.D.*	*X*	*S.D.*
Recovered	27	58.8	±46.2	57.3	±43.0
Improved	42	37.5	±22.8	42.5	±20.2
Unimproved	26	37.2	±24.9	43.1	±23.2

length of the first menstrual cycle in patients classified as recovered and those classified as unimproved was statistically significant at the .05 probability level ($t = 2.10$). The difference between the recovered group and the improved group was also statistically significant at the .05 probability level ($t = 2.18$).

Only the averages of the first cycle during treatment showed these differences in statistically significant proportions. When all cycles during treatment were averaged and the averages compared in the individual groups of improvement, the differences were less significant.

As may be seen from Table I, the mean of the average cycles in the unimproved and the improved group was higher than that of the first cycle, indicating that the second cycle during treatment was longer than the first cycle. In contrast, the mean of the average cycle of the recovered group remained approximately the same as that of the first cycle, indicating that there was a greater tendency for patients classified as recovered to develop amenorrhea early during treatment.

When the schizophrenic patients were classified according to diagnostic subgroups it was found that the length of the cycle was greatest in the hebephrenic subgroup, least in the simple subgroup (Table II).

TABLE II.—Length of First Menstrual Cycle (A) and Average Menstrual Cycle (X) in Relation to Diagnostic Subgroup of Schizophrenia

Subgroup	*Number of Cases*	*A*	*X*
Simple	8	31.4	39.4
Paranoid	19	38.4	44.3
Catatonic	26	40.0	41.6
Other types	27	42.7	47.6
Hebephrenic	15	63.9	62.0
Average	95	43.5	46.9

Only hebephrenic and simple subgroups were found to differ statistically at the .05 probability level. When the diagnostic subgroups were further subdivided according to condition of improvement, the numbers in individual categories became too small for valid statistical comparison. However, the data are presented in Table III since they

TABLE III.—Length of First Menstrual Cycle (A) and Average Menstrual Cycle (X) in Relation to Improvement in Individual Subgroups of Schizophrenia

	Recovered			*Improved*			*Unimproved*		
	Number of Cases	*A*	*X*	*Number of Cases*	*A*	*X*	*Number of Cases*	*A*	*X*
Simple	1	24.0	35.0	5	34.0	36.5	2	28.5	49.0
Paranoid	4	40.3	46.0	11	38.4	44.9	4	36.8	40.8
Catatonic	9	55.3	49.5	11	33.6	38.7	6	28.5	35.2
Other types	10	57.3	54.8	10	38.1	48.3	7	28.3	36.4
Hebephrenic	3	110.7	111.8	5	64.4	56.8	7	43.6	44.4

do show interesting trends which might be corroborated from larger material of other investigators. As may be seen from Table II, there was a consistent increase in the length of the menstrual cycle from the classification unimproved to classification recovered in three diagnostic subgroups, the catatonic, hebephrenic, and other types. In contrast, the paranoid and simple subgroups did not follow this trend. Unfortunately the numbers of recovered patients in the latter two subgroups were insignificant.

Comments

The results of this investigation are difficult to evaluate either from a psychiatric, therapeutic, or endocrinologic aspect. The statistical data confirm the clinical observation of frequent occurrence of amenorrhea during insulin coma treatment. The observation of greater lengthening of the menstrual cycle in schizophrenic patients who benefited most from insulin coma treatment may prove to be an interesting lead for investigation of the mechanism of insulin coma. Paradoxically, the greatest lengthening of the menstrual cycle was seen in the hebephrenic subgroup which is usually considered the least tractable to therapy of any kind.

The compilation in Table III reflects well the established clinical experience with insulin coma. The treatment was clinically most effective in the catatonic subgroup and the subgroup classified "other types," and least beneficial in the paranoid, hebephrenic, and simple types of schizophrenia.

Summary and Conclusions

Insulin coma interferes with the regularity of the menstrual cycle, causing in most instances varying degrees of amenorrhea.

The amenorrhea was significantly greater in patients who were classified recovered, by comparison with all other patients.

The menstrual cycle during coma was longest in the hebephrenic, shortest in the simple type of schizophrenia.

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LAUGHING SPELLS IN PATIENTS, AFTER LOBOTOMY

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Quite a few patients show signs of elation and even euphoria after prefrontal lobotomy was performed on them. It often appears as if the emotions which have been restrained by tension and anxiety suddenly burst out in frolicksome freedom. This first stage of overindulgent facilitation disappears gradually and gives way either to improvement or relapse. It is this emotional facilitation after the operation which raises high hopes for recovery, especially on the part of patient's relatives. Experience has taught us, however, that it has little or no influence on the postoperative course.

Laughing spells after prefrontal lobotomy have not, to my knowledge, as yet been described in the current literature. They present a kind of involuntary laughter which is difficult to interrupt. This paper is concerned with 4 cases who showed these laughing spells to a marked degree.

An article entitled "Fits Of Laughter" (Sham Mirth) in *Organic Cerebral Diseases,* by J. Purdon Martin (5), describes laughing fits which occurred in 4 patients after operations for brain lesions situated close to the hypothalamus. All 4 patients died after the fits of laughter had set in. P. Bard, in his experiments on cats, established the fact that the nervous mechanism for expression of emotions is situated subcortically and is probably connected with the destruction of the posterior nuclei of the hypothalamus. Animals after removal of the neocortex showed pleasurable reactions. Stanley Cobb (2) in "Review of Neuropsychiatry, 1949," stated that by taking away the neocortex but sparing the meso- and archicortex, cats became docile, refractory to nocious stimuli, and somewhat over-ready to react to pleasurable stimulation. The main stimulation seems to come from the hypothalamic hormones, reaching the anterior part of the pituitary by way of the general circulation.

As is well known, patients receiving electric shock treatment become more alert and also show signs of elation. Many of them describe the change in their condition as "waking-up," referring to their former condition as a "dreamlike state," or simply as "having being sleepy all the time." Stanley Cobb (1) says that "physiologically speaking one might speculate that there is a wakefulness center in the hypothalamus

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which keeps the thalamus and the cortex alert. In any type of prefrontal lobotomy the white fibers which connect this lobe with the thalamus are severed. Since all white fibers and not only selected ones are severed, it seems that the connection of the frontal lobe with cortical and subcortical structures are interrupted."

As to the wakefulness center it might be of interest that the laughing spells continue through the night especially at the height of this condition and the patients have to be sedated in order to provide the necessary rest. In some cases even sedation does not suppress the laughing spells completely.

This type of laughing does not show the characteristics of a healthy relaxing laughter; it rather gives the impression of a compulsive explosion, an outburst completely unaccounted for. The maximal laughing spells continue through the first postoperative year. In some cases they subside somewhat later on, though in others they persist almost three years after the operation was performed.

Case Reports

Case 1.—A white female, whose mother was said as having committed suicide during an attack of depression, was born in Germany 36 years ago. Her prepsychotic personality was described as of happy disposition. Her childhood and adolescent years were uneventful. She grew up with a brother and a sister, both her seniors. She finished high school and was considered by her teachers and classmates as kind, efficient, and affectionate. She worked as a typist a few years in Germany before being married in 1937. She had one baby, a son, and entered the United States in 1939. A short time later she was divorced from her husband.

It is said that she was once before hospitalized for a short time in Neuchâtel, Switzerland. Early in 1946 she became depressed and developed delusions of persecutions about the food being poisoned and the child not being her own. She was admitted the same year to a mental institution because she became confused, unco-operative, tense, agitated, incoherent, and irrelevant. She received 69 electric shock treatments and insulin treatment, but continued to be negativistic, withdrawn, and finally became completely mute and a feeding problem, with periods of excitement and assaultiveness. In the course of her disease she also showed waxy flexibility, rigidity and spastic stiffness of the legs. She received electric shock treatment on a symptomatic basis without any further benefit. The diagnosis was dementia praecox, catatonic type.

In April 1949, a bilateral prefrontal lobotomy was performed and a short time later she became overactive and overtalkative, elated and exuberant to the point of annoyance. She also developed laughing mirth, laughing constantly in a loud and noisy manner. Her laughing seemed to be a kind of compulsion which she was unable to control. She had to be separated from the other patients whom she disturbed greatly by her uncontrollable

laughter. She admitted being unhappy because she had to stay in the hospital but was unable to control her laughing fits.

The type of laughing in this case was like a stream of constant merriment. It was as if she were overflowing in enjoyable amusement, yet there was nothing contagious in her laughter, which annoyed patients and employees alike. Her memory except for the time of her mental disease is fair; she is fairly alert, able to work but prevented from doing so because of her laughing spells.

Case 2.—A 40-year-old, native-born white female patient. Her birth and early development did not show any unusual deviations from the norm. She was said to have been bright in school, finished high school, and attended business school. She was never seriously ill but felt that being born a Jewess hindered her advancement. She showed the first signs of her psychosis at the age of 22 when she became depressed, confused, and turned against her mother. She refused to eat, could not sleep and was admitted to a private sanatorium where she remained for nine months, and was discharged against advice. She was diagnosed as manic depressive psychosis, manic type, and though she never entirely recovered she held a job for three years. During the following years she was unable to hold a position for a longer time and changed from job to job. At the age of 29 she fell in love with a young man and became engaged to him after a short acquaintance. A few days prior to their wedding the fiancé discovered that she had been hospitalized previously and broke off the engagement. The patient reacted with great excitement and restlessness, became irrational and violent, and was admitted to a state hospital where she was diagnosed as dementia praecox, hebephrenic type. She improved gradually, was released from the hospital after six months and discharged after one year. About two years after her discharge, she again showed active psychotic symptoms, became withdrawn, suspicious, and aggressive toward the neighbors. She was given electric shock treatment without benefit.

In June 1949, a bilateral prefrontal lobotomy was performed and after a few weeks she became more alert, spontaneous, quiet, and pleasant. Six weeks later she was discharged and entered a private nursing home. It was there that she developed screaming spells, again became restless and overactive, and was admitted to Pilgrim State Hospital. On admission she shouted and screamed at the top of her voice, was unco-operative and agitated but soon showed behavior improvement. She was quiet and co-operative but developed a kind of laughter which came on in spells and which she evidently was unable to suppress. At first she would laugh incessantly and for this reason had to be separated from the other patients. She could not stop on her own volition or on command. She did not react to medication. In February she went into a status epilepticus but reacted well to anticonvulsive drugs, and has not had any seizures since. Her laughing spells are about "a dozen times a day," according to the patient's own statement. She now stops on command only to burst out again a short while later. Her general condition has improved, she is helping with work and does it fairly

efficiently. She is friendly and gets along well with the other patients. Hers is a kind of laughter best described as a "frozen laughter." A shrill, harsh noise is prevalent in her laughing spells. Her face is distorted and tense rather than relaxed. Asked for the cause of her laughing she states that she "sees people crawling around on the floor."

Case 3.—The mother of this patient was admitted to a state hospital because of a psychosis due to arteriosclerosis. She was hospitalized three months, discharged two years later, and died a short time afterward. The patient is at present 50 years of age, an Italian-born white female. She had little schooling, did farm work and was married at the age of 21. She gave birth to three children who all are well established in life. Her prepsychotic personality is described as simple, but quiet, cheerful, and happy. She was a good wife and mother, affectionate toward her family who always was in adequate financial conditions. She was religious, but became over-religious prior to the onset of her psychosis spending nights in prayer and days in church. When she was about 39, her husband injured a finger and had to be hospitalized. The patient developed the idea that someone would kill her husband and she attempted suicide by drinking iodine. She was hospitalized and transferred to a mental hospital because she became agitated, restless, and so apprehensive that she would run and hide when even a member of the family would enter the room. She insisted that the devil was coming into the room and was obviously actively hallucinated. She stated that "she was scared to death," and was afraid that the "injections would drive her crazy." She exhibited a childish behavior, was uncertain, and screamed if one came near her. At other times she would sing in a loud voice and behave in such a childlike manner that she was diagnosed "psychosis with mental deficiency." In the hospital she would play with all kind of trash, sitting on the floor mumbling to herself, and adorning herself in a childish fashion. In April 1947, she was transferred to Pilgrim State Hospital where she had to be kept in a disturbed ward because of her noisy and destructive behavior. She received electric shock treatment with little improvement. She was diagnosed as dementia praecox, hebephrenic type.

In February 1951, a bilateral prefrontal lobotomy was performed. Following the operation, she showed improvement in behavior, became much less tense, agitated, and fearful, but developed a kind of compulsive laughter, bursting out without any apparent reason at the most inconvenient times. Though these laughing spells have subsided to a certain extent, they still persist and she is unable to give any reason for them. They come on at irregular intervals and resemble the pleasurable laughter of a playful child, though they do not induce others to join in them.

Case 4.—A 33-year-old white American female whose mother committed suicide while the patient was hospitalized. A paternal aunt was feeble-minded and epileptic. The patient's early development was normal. She was operated on at the age of six because of intestinal obstruction but

recovered without complications. During her school years the family moved around quite frequently and the patient was unable to settle down for a long time. She was a good student and received many scholastic honors. In spite of going out with boys and girls, she always was rather retiring. During her second college year, she was under the care of a psychiatrist because she felt she had trouble with her work and could not concentrate. Psychotherapy set her right but she became even more retiring than before. After graduation from college, she became a saleslady making selling her career. She became engaged when she was 20 but this engagement was later broken off. About a year later, after having attended the movie "Lady In The Dark," she became emotionally upset, uttered ideas of de-realization and de-personalization, and was admitted to a private sanatorium. There she was described as preoccupied, neglecting herself. Insulin treatment was applied which helped her to leave the hospital after eight months. She seemed recovered, worked steadily until 1943 when she married. In 1944 she gave birth to her first baby and from then on moved around following her husband who was called into the Army. When he went overseas, she returned to live with her parents. After her husband returned home, they settled down and she gave birth to her second child. A few months later she became upset, admitted hearing voices which frightened her. She was admitted to a hospital, received electric shock treatment and was discharged. In 1949 she was admitted to Pilgrim State Hospital because she was disturbed, actively hallucinated, and had developed paranoid ideas against her husband. In the hospital she became assaultive, unpredictable, and a severe feeding problem. She was diagnosed as dementia praecox, catatonic type, and in September 1950 a bilateral prefrontal lobotomy was performed. The postoperative course was uneventful, she became more tidy and co-operative, ate and slept well but still seemed preoccupied and delusional. A few months after the operation she developed fits of laughter which she could not suppress. At the maximum of her laughing spells, she did not respond to medication and had to be kept away from other patients whom she disturbed by her laughing spells day and night. She laughs with a "dead pan" face and cannot give any explanation for the laughter but says, "I know it is stupid and I try to control myself; maybe I relax too much." She does not seem to derive any pleasure from her laughing and in group therapy sessions she will suddenly burst out without any apparent reason. None of the other patients join into her laughter, they rather seemed to be annoyed and disturbed by it.

There seem to be no meaning in the laughing spells just described. The 4 patients laugh as if all inhibitory mechanisms have been released at once. Their laughter is completely meaningless and not contagious. It sometimes seems as if they would rather cry than laugh. Their facial expression is distorted and tense and not relaxed.

George A. Colom and Morris Levine have reported a case of self-inflicted lobotomy by a gunshot wound. They state that the loss of a

previous depression and the postoperative euphoria were the most impressive symptoms, together with childishness, loss of initiative, facetiousness, and extroversion. Looking for the cerebral central mechanism, E. A. Spiegel and H. T. Wyce found that it must be sought in the subcortex, cranial to the midbrain. "Emotions cannot be referred to a single circumscribed nucleus within the diencephalon or to its connection with the frontal lobes. There exist a multiple representation of emotional function."

All of these experiments and theories emphasize the fact that the organic mechanism of emotions is a rather complex one but most probably intimately connected with the diencephalon and its cortical connections. This may account for the variety of results achieved by prefrontal lobotomy. In studying a number of cases we soon become aware that generalizations such as change in personality, childlike regression, loss of initiative, and spontaneity may refer to some of the cases but certainly not to all of them. Not even the least unquestionable result achieved by prefrontal lobotomy such as facilitation of tension and anxiety are unfailing results in all cases. In some tension, depression, anxiety, and agitation persist. This is true for the phasic course of some schizophrenic reaction types which usually present a favorable indication for prefrontal lobotomy.

Symptoms may be obliterated by the operation only to return later on, and not even the duration of the illness is indicative of the outcome of the operation. Some of the best results were achieved in cases of long standing. There may be some truth in the German concept that the age at onset of symptoms is the most important factor in predicting the outcome of treatment. The older the patient when the symptoms develop, the more favorable may be the result of any treatment.

Inasmuch as prefrontal lobotomy releases patients of the straight jacket of inhibition, it may also cause uninhibition to such an extent that previously controlled functions may become disorganized and released to the extreme. This is one theoretical consideration in cases with laughing spells after prefrontal lobotomy.

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MINNESOTA MULTIPHASIC PERSONALITY INVENTORY PATTERNS BEFORE AND AFTER CARBON DIOXIDE INHALATION THERAPY*

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Carbon dioxide inhalation therapy was introduced by Meduna in 1947 as a treatment for neuroses and allied conditions. The usefulness of this therapy has remained controversial and inconclusive, although many thousands of individual treatments have been administered. In a series of 100 patients with diagnoses of anxiety state, hysteria, spastic colitis, and stuttering, Meduna claimed relief of symptoms in 68 per cent (3). He uses a purely pharmacologic approach, with no psychotherapeutic interviews, and believes that the biochemical effects of the gas have theraputic value. Wilcox (7) combines carbon dioxide treatment with psychotherapy, and while he considers the neurophysiologic reactions important, feels that psychodynamic changes are facilitated making it possible to uncover unconscious material which may result in insight and subsequent clinical improvement. Kindwall (1) compares carbon dioxide therapy to a brief, elective, controllable toxic psychosis, and reports favorable results, mainly in alcoholism, barbiturate addiction, anxiety states, and psychosomatic conditions. Carbon dioxide inhalations have been used by Liest (2) in the treatment of schizophrenic patients in partial remission. He observed that previously dynamically sterile patients began recalling long-repressed material of a traumatic nature. Moriarty (4) concludes that while carbon dioxide therapy alone is beneficial, improvement is enhanced by correlated psychotherapeutic interviews. He calls attention to his experience with patients whose illness had been resistant to psychotherapy but responded favorably when carbon dioxide inhalations were added to their treatment program. He reports that in a series of 66 patients 39 per cent were much improved, 43 per cent improved, and 18 per cent unimproved. A favorable response was obtained in anxiety states, phobic

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reactions, some types of character neurosis, chronic tension states, psychosomatic conditions, and alcoholism with prominent psychoneurotic factors. Patients with conversion hysteria, or with fairly pronounced depressive or schizoid features, reacted less favorably to carbon dioxide therapy. He states that this treatment is of no lasting benefit in the major psychoses and in obsessive-compulsive states. He also observed that carbon dioxide produces an abreaction which is more effective, and is safer to administer than sodium pentathol or ether, and that the patient is much more accessible and receptive to interpretations immediately following an inhalation treatment.

The purpose of the present study is to further explore some of these observations through the use of the Minnesota Multiphasic Personality Inventory (MMPI).

Method

1. A group of 38 male patients who had received carbon dioxide therapy in addition to psychotherapy during their hospitalization were selected for study. This group was compared with a control group of 38 patients who were closely matched in terms of diagnosis, but who had not received carbon dioxide therapy. The two groups were tested with the MMPI, both before and after treatment, and compared in terms of their mean profiles. The diagnostic composition of the groups was 23 anxiety reactions, 8 psychophysiologic reactions, 4 depressive reactions, 2 character disorders, and 1 phobic reaction. The mean age of the carbon dioxide therapy group was 33.4 years, with a range of 24-48. The mean age of the control group was 34.1, with a range of 26-58. Both groups had an average hospitalization period of 56 days.

2. The total available sample of carbon dioxide treated patients who had received the MMPI both before and after treatment consisted of 45 patients. (Seven patients were omitted from the above controlled study because of difficulty in matching.) These 45 patients were divided into two groups: (a) those responding favorably, and (b) those responding unfavorably to carbon dioxide therapy, as determined from the discharge summaries. The two groups were compared also in terms of their mean MMPI profiles before and after treatment. Twenty-five patients, or 56 per cent, fell into the favorable response group, and 20 patients, or 44 per cent, into the unfavorable response group.

Results

Figure 1 contrasts the mean profiles before treatment of the carbon dioxide therapy group and the control group. There are no significant differences on any of the scales. (The t test of the differences between the means of correlated measures was used.) The shape of the profiles

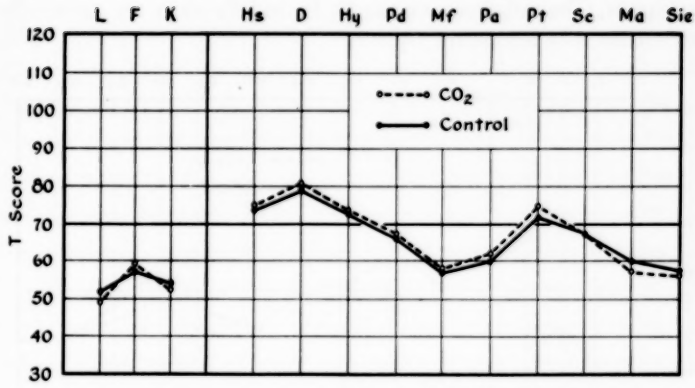

FIG. 1.—Pre-treatment mean profiles of CO_2 and control groups.

is typical of mean profiles reported for hospitalized male psychoneurotic patients (5).

Figure 2 contrasts the mean profiles after treatment of the carbon dioxide therapy group and the control group. Again, there are no

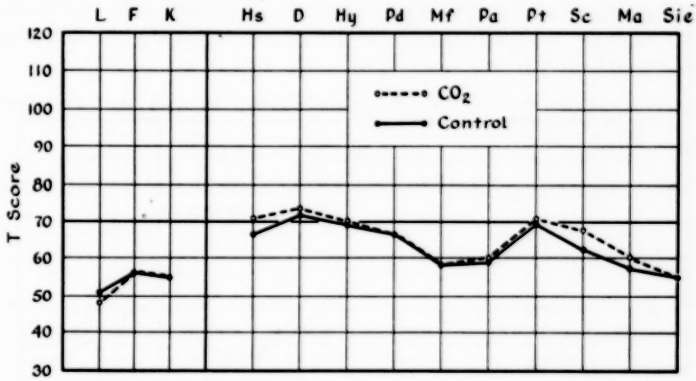

FIG. 2.—Post-treatment mean profiles of CO_2 and control groups.

statistically significant differences on any of the scales. Both groups have lower mean profiles following treatment (Fig. 2 contrasted with Fig. 1), but Fig. 2 does not suggest any marked differential effect on the MMPI mean profiles resulting from the additional factor of carbon dioxide therapy.

Figure 3 contrasts the mean profiles before and after carbon dioxide treatment of the 25 patients in the favorable response group. It will be seen that all of the scales except K are either lower or unchanged after

treatment. The differences on the D, Pt, and Pa scales are statistically significant at or beyond the .05 level.

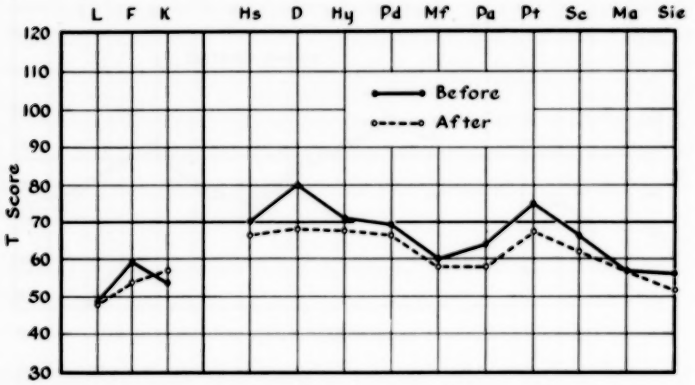

FIG. 3.—Mean profiles of the favorable response group before and after CO_2 therapy.

Figure 4 contrasts the mean profiles before and after carbon dioxide treatment of the 20 patients in the unfavorable response group. In this case all of the scales are either higher or unchanged after treatment. The changes on the Pd, Sc, and Ma scales are statistically significant at or beyond the .05 level.

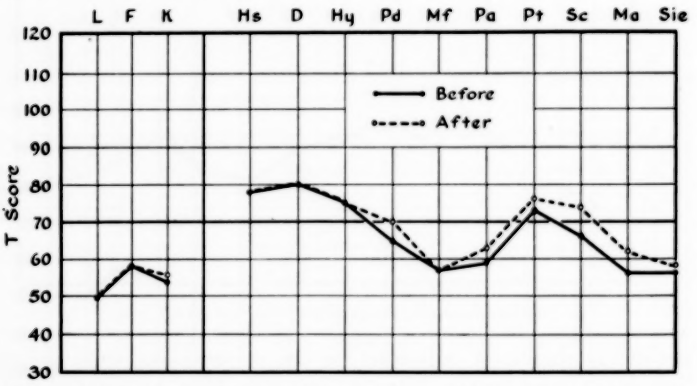

FIG. 4.—Mean profiles of the unfavorable response group before and after CO_2 therapy.

Figure 5 contrasts the mean profiles of the favorable and unfavorable response groups before carbon dioxide treatment. Although none of the differences reaches the .05 level of statistical significance, there appears to be a trend for the Hs and Hy scales to be higher, and the Pa scale to be lower, in the unimproved group.

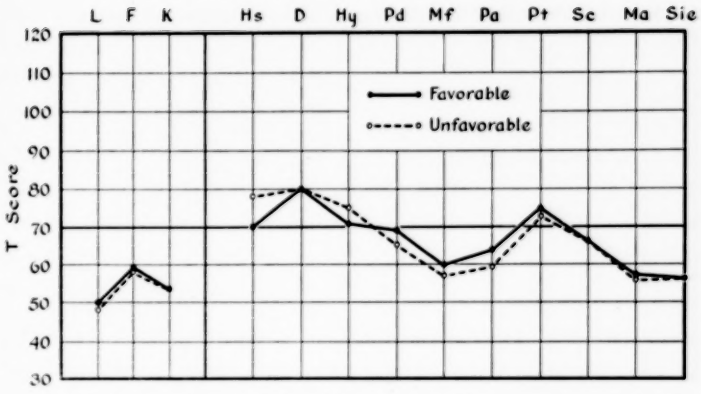

FIG. 5.—Mean profiles of the favorable and unfavorable response groups before CO_2 therapy.

DISCUSSION

It is probably no longer necessary to discuss at length the usefulness of the MMPI in evaluating and measuring effects of various therapies. Schofield (5) concludes that: "The MMPI profiles of successfully treated psychiatric patients reflect the success of therapy in significantly reduced scale scores. Where the success of therapy is less definite and where there is reason to surmise lack of improvement because of the nature and extent of therapy, the age and intelligence of the patients, and other factors, the MMPI scale scores remain essentially unchanged." West (6) also demonstrates how the MMPI can be used in measurement of changing psychopathology.

Moriarty (4) has observed that many patients who are not accessible to psychotherapy become more responsive with carbon dioxide treatments in addition to psychotherapy. This observation is supported by our own clinical experience over the past 18 months. Patients who respond favorably to carbon dioxide therapy appear to become more readily available for psychotherapy, as indicated by (a) repeated ventilation of problems in the form of abreaction and dreams; (b) ability to face problems without excessive anxiety; (c) apparent increase of self-confidence; and (d) increased ability to control impulsive behavior.

The MMPI data would seem to indicate that as a group the patients treated with carbon dioxide and psychotherapy are little different after treatment from a group of psychoneurotic patients treated with psychotherapy but not receiving carbon dioxide. There was no difference in the length of hospitalization for the two groups (56 days). From these findings it might be argued that the addition of carbon dioxide to the

treatment regime was of little value. However, if one takes into consideration the fact that the patients to receive carbon dioxide were initially selected because of their resistance and inaccessibility to psychotherapy, one might conclude that the same beneficial results were obtained with a more difficult group of patients in the same length of time. We do not feel that our data answer this question beyond the empirical nature of the MMPI findings.

Moriarty (4) has also observed that individuals with marked schizoid and conversion features do not tend to respond favorably to carbon dioxide therapy. These observations appear to be supported by our data. The unfavorable response group in Fig. 4 failed to show any lowering of the scales reflecting anxiety (D and Pt) after treatment, but did show a significant rise on scales reflecting schizoid trends (Sc) and restlessness (Pd plus Ma). Since these differences were not particularly apparent before treatment (Fig. 5) it might be hypothesized that the schizoid trends were latent or subclinical and were brought to the surface and made manifest by the carbon dioxide treatment. The trend toward higher Hs and Hy scores of the unfavorable response group (Fig. 5) is consistent with Moriarty's observation that patients with conversion hysteria do not respond well to carbon dioxide therapy. It is our clinical impression that patients who do respond well to carbon dioxide therapy are more often characterized by "free-floating" anxiety. This empiric impression appears to be supported by the MMPI data in Fig. 3, which shows elevation of the anxiety indicators (D and Pt) prior to treatment, and statistically significant decrease of these scales after treatment.

A number of questions remain unanswered in an attempt to completely evaluate the effectiveness of carbon dioxide therapy. Without dwelling on the extensive physiologic alterations in the internal environment which this form of treatment produces, we have been impressed with the marked relief of overt anxiety following each inhalation treatment, which tends to become of longer duration in the intervals between treatments. However, the clinical observation which appears to be most characteristic is that patients originally unavailable for psychotherapy, apparently because of their defense mechanisms, become capable of facing their problems during carbon dioxide therapy. Since this is based entirely on clinical impression and judgment, it would seem desirable to find proof for this hypothesis in a future project.

Summary

1. Thirty-eight male patients who received carbon dioxide therapy and psychotherapy were compared with a closely matched group of

38 patients who received only psychotherapy. Both groups were tested with the MMPI before and after treatment and their mean profiles were compared.

2. No statistically significant differences were found in the MMPI before and after treatment in the carbon dioxide therapy group and the control group.

3. Forty-five male patients who were treated with carbon dioxide therapy and psychotherapy were divided into improved and unimproved groups and their mean MMPI profiles before and after treatment compared.

4. The favorable response group reveals statistically significant decreases in the D, Pt, and Pa scales of the MMPI, indicating decrease in anxiety.

5. The unfavorable response group shows all scales of the MMPI unchanged or higher after treatment. The changes on the Pd, Sc and Ma scales are statistically significant, indicating increased restlessness and schizoid trends.

6. Comparison between the mean MMPI profiles of the favorable and unfavorable response group before carbon dioxide therapy shows no differences of statistical significance, but a trend for the Hs and Hy scales to be higher and the Pa scale to be lower in the unimproved group.

7. The observation that patients with schizoid and hysteroid features tend not to respond favorably to carbon dioxide therapy appears to be supported by our data. However, patients with "free-floating" anxiety, as indicated by the MMPI, do respond favorably.

8. The hypothesis that patients, resistant to psychotherapy because of their defenses, become more readily accessible to psychotherapy through the addition of carbon dioxide therapy, is discussed, but these clinical observations are not reflected in our MMPI profiles.

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(Numbers of references changed in text in accordance with alphabetized listing.)

DURATION OF CONVULSION IN ELECTRIC SHOCK THERAPY *

RICHARD W. FINNER, M.D.†

The Problem and Experimental Procedure

Review of the American medical literature and examination of some foreign reports reveal gaps in our knowledge concerning the length of convulsion experienced by patients receiving electric shock therapy (EST). Several experimenters have indicated values ranging from 23 to 93 seconds for the shortest and longest convulsions observed; the average is generally estimated at 40 to 45 seconds. Different types and amounts of electrical stimulation have been used, and other factors that may influence the length of convulsion have usually not been controlled. Noticeably lacking in these reports is correlation of the length of convulsion with other factors about the individual patients. To gain information on questions raised, an experiment was designed to study length of convulsion in patients receiving EST, and to attempt correlation of this time period with other factors about the patients, such as sex, age, or nature of illness.

Observations were made on 39 patients undergoing a course of EST in the In-Patient Department of the Department of Psychiatry and Mental Hygiene, during the winter of 1946-47. Treatments were given three times a week before breakfast.

The instrument‡ used was so calibrated that any current from 200 to 800 milliamperes could be given; 350 ma. was the usual and minimum amount used. Time of administration could be varied in regular steps from 0.05 of a second to 1.0 second. The usual and minimum time was 0.3 of a second; 110 volt, 60 cycle, AC was employed. Small metal electrodes were used; these were held in place by hand, one over the left temporal region and the other over the occiput. Electrode jelly was employed. Time measurements were made with a manually operated stop watch; the time recorded was that of the nearest complete second. The watch was started at the moment the current was administered to the patient—then started again if a second or third stimulus

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‡ Made by Offner Electronics, Inc., Chicago, labeled Electric Shock Therapy Apparatus, Type 732, Serial 257.

was necessary. The times of atypical seizures and "subconvulsive responses" were not included in this study. Cessation of the clonic phase of the major convulsions was taken as the end of the time period; this was usually in advance of the first postconvulsive breath. At times, some difficulty was encountered in determining the exact end of a seizure, and it was occasionally necessary to add or subtract an estimated three to five seconds from the period recorded by the watch. Various isolated postconvulsive movements, not actually part of the generalized seizure, were not included. In the present study no attempt was made to time the latent period—the interval between administration of the current and onset of the seizure; it was brief or nonexistent. This fact is mentioned only because it may be of importance in comparing the present data with those obtained by others.

Preliminary Results

In the present experiment there were 10 men and 29 women, and there were 317 timed convulsions. Some patients had only a few convulsions; others had 20 or more. The range of the distribution was from 25 to 57 seconds, with a mean of 41.38 seconds, and a standard deviation of ± 5.52. Both median and mode were 41 seconds. These calculations are based on the entire uncorrected array of scores and they give only a preliminary view of the study. At this stage of the investigation, several factors that may affect the length of convulsion are not controlled, and, consequently, some scores must be eliminated in order that valid comparisons may be made. The nature of these corrections will be discussed in the next section.

It may be mentioned here that the figure for the mean (41.38) compares favorably with reports of others in the literature. Sulzbach and coauthors reported from uncorrected scores a mean of 39.9 seconds; this includes their estimate of the latency period, 1.8 seconds, which they timed as a separate period. It is also of interest that the range in the present study is almost identical with that of Sulzbach and associates, who reported a range of 24.8 to 56.8; the time of the laent period is again included.

Corrections for Factors That Might Influence the Measurements

Selection of scores for comparison was made on the basis of the following objective criteria.

In the interest of uniformity of conditions, all time periods of single stimuli longer than 0.3 sec. were excluded. Twelve convulsions were thus eliminated. Next, in women, convulsions which occurred as a result of one shock were compared with those which occurred as a

result of two or more shocks; sufficient significant differences were found to eliminate convulsions in which two or more shocks were used. It was next ascertained that in women no correction was necessary for the amount of amperage used to obtain a convulsion.

To check for the experimenter's error, the length of the first convulsion of each woman who started treatment in December, January, and February was compared, month with month. No significant differences were found, and consequently the records for each month were regarded as independently valid. The reaction time of the observer may possibly play a part in highly refined studies. In the present case, this was 0.22 sec. for visual stimulation; this figure was neglected in the present study.

A comparison of the convulsion lengths in men with those of women yielded inconclusive results, and for the major purposes of the study the data concerning men were omitted, since the data from women were more extensive.

No patients were included in the principal comparisons who had had EST within one month (an arbitrary standard) before their present therapy began.

Finally, data were further restricted so that the main inquiry is based on the records of 12 women, each of whom had 8 convulsions. Women who had less than 8 convulsions were not included, and where more than 8 had been timed, only the first 8 convulsions were used. The corrections for multiple shocks, etc., necessitated omitting a few convulsions within this select group. However, since the mean of the performance of each patient is used, only small numerical differences enter. Data concerning the 12 patients are condensed in Table I.

TABLE I.—Data Concerning the Selected Group of 12 Women Receiving EST

Pt.	*Age (Yr.)*	*Dx.*	*Immediate Result of EST*	*Duration*	*Prev. EST*	*Wt. (Kg.)*	*Ht. (Cm.)*	*Body Area (Sq. Me.)*	*Mean Sec.*	*S.D.*
1.	23	S	Good	Indef.	No	55.2	161	1.57	48	3.46
2.	45	S	Good	Indef.	Yes	54.3	166	1.59	47	4.10
3.	25	N	Poor	Indef.	No	60.6	164	1.66	46	2.79
4.	66	N	Good	Indef.	No	53.3	158	1.53	46	2.89
5.	36	S	Poor	<1 mo.	No	48.3	165	1.51	45	2.72
6.	25	S	Poor	>6 mo.	Yes	61.8	169	1.71	44	2.03
7.	37	S	Good	Indef.	No	60.8	170	1.71	42	2.00
8.	18	S	Good	>6 mo.	No	52.0	168	1.58	41	1.51
9.	27	S	Good	<1 mo.	No	48.7	159	1.48	40	4.30
10.	33	S	Good	<1 mo.	No	41.2	150	1.32	39	1.90
11.	21	S	Poor	>6 mo.	Yes	55.3	164	1.59	38	2.52
12.	25	S	Good	Indef.	No	45.5	159	1.44	32	2.27

Explanation of Table I:
Dx. indicates diagnosis, *S*—schizophrenic; *N*—nonschizophrenic.
Duration refers to duration of illness before treatment.
Prev. EST indicates patients (3) who had EST within one year before starting present therapy. No patient included who had EST within one month of present treatment.
Mean Sec. indicates mean length of convulsion in seconds for the first 8 convulsions of each patient.
S.D.—standard deviation.

The problem of the control of other variables in the personal history and clinical picture of the patients is a research project in itself, and was not feasible in this experiment. This fact limited the number of comparisons to those reported in the next section.

Results of Investigation

1. *General Character of Performance.* The mean and standard deviation of the performance of each patient in the select group of 12 may be noted in Table I. Means vary from 32 to 48, and the fact of individual differences in the means of subjects is clearly shown. The significance of both the mean and the standard deviation, in relation to various problems manifested by each patient, requires separate study.

2. *Comparison of Mean Length of Convulsions in a Series.* In the

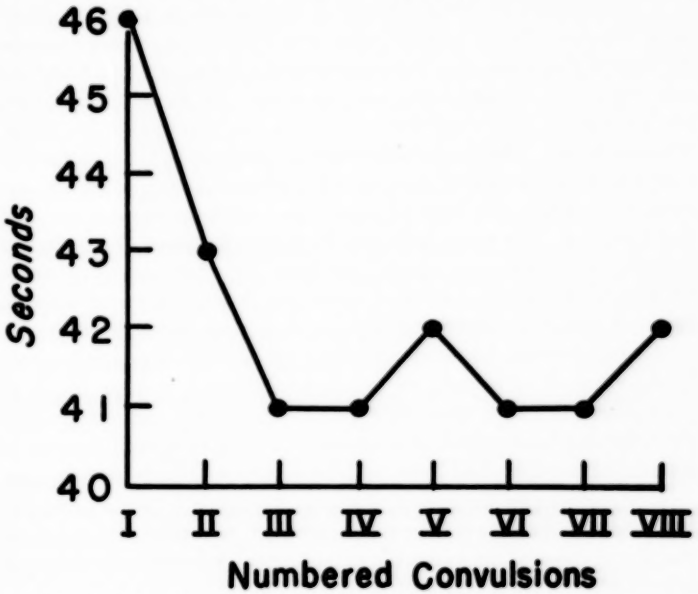

Fig. 1.—Graph showing the mean length of convulsion for the series of eight different convulsions. The numbers on the abscissa indicate the number of each convulsion in its own series; the mean of first convulsions is significantly longer than that of any other.

Acknowledgment: To Dr. Stanley T. Michael, formerly instructor in the Department of Psychiatry and Mental Hygiene, Yale University School of Medicine, I am indebted for much helpful criticism and the suggestion that studies be made on the length of convulsion experienced by patients in electric shock therapy. I am also grateful to Dr. John Richard Wittenborn, Research Associate in Psychology, and to Dr. George F. Mahl, Associate Professor of Psychiatry (Psychology), Yale University, for advice on the statistical methods of analysis.

group of 12 patients, the next problem considered is the comparative length of different numbered convulsions—1 through 8—in each series. Figure 1 shows the mean for each of the eight groups of seizures. The mean of first convulsions, 46 seconds, is significantly longer than that of any other. Also, when the mean of first convulsions is compared with the mean of all others, by use of the *t* test, a value of 3.15 is obtained; this indicates that the figures are significant at about the 1 per cent level. Although the mean of second convulsions is not significantly different from the others that follow, it may be noted in passing that it is larger than all other except the first. From the fact that the length of convulsion becomes shorter after the first seizure in a series, in women, it may possibly be concluded that some form of adaptation occurs during EST.

3. *Comparison of Mean Length of Convulsion in Different Diagnostic Groups.* The 12 patients were next divided into two groups on the basis of diagnosis. Ten had schizophrenia, the simple type being the only one of the classical forms not represented. Of the other 2 patients, one had a severe psychoneurosis and the other a depression with cerebral arteriorsclerosis. The 2 nonschizophrenic patients had a significantly longer time of convulsion than the schizophrenic group, 46 and 42 seconds, respectively, the level of significance being between 1 per cent and 5 per cent. This seems to fit well Hoskins' conception of a "defective reactivity to stimulating agents" exhibited by schizophrenic patients. Because of the small number of cases, any conclusion remains speculative; it would be interesting to have more data on this matter.

4. *Miscellaneous Comparisons within the Selected Group of Patients.* In the group of 12 women, no significant difference in mean length of convulsion was found when the patients were divided and compared on the following bases: result of treatment, duration of illness, presence or absence of previous EST, age, weight, height and body surface area.

5. *Miscellaneous Comparisons within the Total Group of Patients.* Inspection of the data for the entire group of 39 patients did not show that the number of days (usually two or three) between convulsions in a series had any effect on length of convulsion.

On inspection, there was no evidence that a sedative given the night before treatment had any effect on length of convulsion.

In the group of 29 women, 18 convulsions occurred during the menstrual period or within two days of its onset. On inspection, no apparent difference in length was found between these and other convulsions.

It will be recalled that in women there was a significant difference in length of convulsion, dependent on whether one or more shocks had been given. The longer convulsions occurred as a result of single

shocks; this finding is in agreement with the previous report of Hemphill and Walter who also made this observation in women. A similar comparison made with the records of men again revealed significant differences. The important point, however, is that wherever a significant difference was found, the longer mean occurred with the convulsions elicited by two or more shocks. More data are necessary, it is believed, before conclusions regarding this apparent difference in the sexes can be drawn. It is possible that the amount of time between repeated shocks might bear on this matter. Occasionally, this interval was as long as 30 seconds, but the period was not regularly timed.

Data were insufficient to show whether there is a difference in length of convulsion between men and women.

6. *Observations on Threshold for EST.* Although the work was not designed to study threshold values, a few general observations are reported. The literature contains many studies concerning such values. In our experience the threshold for EST tends to rise as a series of convulsions progresses; this is in agreement with the reports of others. Watterson found a positive correlation in men between threshold for convulsion and age, and Kalinowsky and Hoch also mentioned that the threshold is higher in middle-aged than in younger individuals. Inspection of our data likewise indicates that a higher threshold is more apt to be found in older patients. The present data do not support the statement of Kalinowsky and Hoch that men have a lower threshold for EST than do women, although only a few convulsions can be compared and the results in this respect must be interpreted with caution.

7. *Comments on Adaptation Occurring in EST.* The elevated seizure threshold observed as a series of convulsions progresses, and the fact that in women convulsions become shorter after the first convulsion in a series suggest that adaptation occurs in patients receiving EST. The adaptive response may be related to that which accounts for the increased seizure threshold noted in older patients; further speculation on the nature of the adaptive process is not warranted from the present data.

Summary and Conclusions

1. Observations were made on length of convulsion in 39 patients receiving EST. There were 317 timed convulsions; the uncorrected measures gave a mean of 41.38 seconds with a standard deviation of ± 5.52. These figures compare favorably with reports of others in the literature.

2. A group of 12 women—each of whom had had at least eight convulsions—was selected for making certain comparisons. The range

in mean length of convulsion for the 12 patients was from 32 to 48 seconds.

3. For the selected group, the mean of first convulsions was significantly longer than the mean of any other numbered convulsion, for the series of 8.

4. Ten women with schizophrenia had convulsions which were significantly shorter than those of two women with other illnesses. The meaning of this fact may possibly be explained in terms of the "defective reactivity to stimulating agents" of schizophrenic patients.

5. No significant difference in mean length of convulsion was found in the group of 12 women when they were divided and compared on the following bases: outcome of treatment, duration of illness, presence or absence of previous EST, age, weight, height and body surface area.

6. There is no evidence that the number of days elapsed between treatments in a series, sedation given the night before treatment, or menstruation have any effect on length of convulsion.

7. The results of this study agree with previous reports that the length of convulsion in women is shortened when two or more shocks are administered. This was not demonstrated in men. It is possible that the number of seconds elapsing between successive shocks may affect the duration of convulsion.

8. Data were insufficient to show whether there is a difference in length of convulsion between men and women.

9. Inspection of the data shows that threshold values for EST tend to rise as a series of convulsions progresses; also, that a higher threshold is more apt to be found in older patients. This is in agreement with reports of others in the literature. The data in the present study do not support previous observations that men have a lower threshold for EST than do women.

10. Evidence for an adaptive process occurring in patients receiving EST is presented. Seizure thresholds are elevated as a series of treatments progresses, and, in women, the first convulsion in a series is significantly longer than those that follow. The nature of the adaptive process is not known. It may be related to the observation that seizure thresholds increase with the age of the patient.

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OBSERVATIONS ON SHOCK THERAPY

Selected Direct Cerebral Intracranial Electroshock Therapy

JUAN NEGRIN, JR.,* M.D.

A general survey on methods at our disposal to treat mental illness shows that we have, aside from psychotherapy and analysis, a group of medical procedures of which the one most widely used is electroshock. During the past few years there have been many efforts to improve the therapeutic effect of electric shock. With this purpose in mind the electric current has been supposedly directed toward specific brain regions by varying the site, place of application, shape and size and materials of the electrodes used and, also, by changing the type of electric current.

On the other hand, we have a group of more radical techniques included in that part of neurologic surgery, known as psychosurgery, which treat mental illness by way of operations on the brain hemisphere.

The failures to simple electroshock therapy or combined with pharmacological shocks are many. The specific indications of a particular psychosurgical technique are not yet clearly defined and the results cannot be predicted.

We have missed a method to treat psychosis which is, as far as its radical nature, somewhere between the electric shock and the usual psychosurgical procedures. Several years ago I thought that the administration of electric shock to the brain by way of intracranially placed electrodes would improve the therapeutic results of electric shock because of its more selective and direct application to specific cerebral regions. Although it is a more radical type of treatment than the external electric shock, it would not produce the extensive irreversible anatomic changes of the psychosurgical technique commonly employed.

Prior to 1947, several types of experimental electrodes were used. In 1948, a type of electrode was obtained, built according to specifications, with the co-operation of Medcraft, which permitted the start of this project in clinical material.

METHOD

1. Trephine opening in the usual manner, over the area selected for electrode placements.

2. A small channel is rongeured in the bone removed, to allow

* From the Neurosurgical Services of Bellevue and Lenox Hill Hospitals.

FIG. 1.—X-ray appearance of the intracranial electrodes (Model M. H. 26) placed over selected brain areas for direct cerebral electroshock therapy.

the connecting extension from the electrode to reach the surface of the scalp.

3. The electrodes are applied to the chosen cerebral (cortical or parenchymal) areas, over one or both hemispheres.

4. The dura is replaced to its preoperative position.

5. The bone disc is replaced.

6. Closure in layers follows.

7. A dry dressing is applied. A head plaster cast can be added should the patient's condition and lack of co-operation indicate it.

8. At continuation, the electric shock treatments are given by connecting the wire of the electrode to the source of the electric current. The patient can be ambulatory in between each treatment if his clinical condition permits.

9. When an adequate number of treatments is given, the wound is reopened and the electrodes removed. The electrodes have been left in place over 14 weeks, without untoward reaction. We are now working on an electrode which can be removed without re-operation.

Case Report

In order to illustrate the technique used, and to show the results of this type of treatment the case of a fifty-three-year-old female patient is presented. Her history and clinical findings were consistent with a chronic psychoneurosis. At the age of thirty-four she fell downstairs and sustained a sacroiliac injury followed by constant low back pain. Two and a half years later there was weakness of the right leg.

Her complaints increased gradually and she had a negative exploratory laminectomy five years after her fall. After this operation, she developed paralysis of the left lower extremity and a severe paresis of the right lower extremity. Four years later a re-exploration of the laminectomy demonstrated extensive piarachnoiditis. After this operation there was no improvement; instead, excruciating constant pain of the left lower extremity appeared. A lumbar sympathectomy was done, at the age of 47, to relieve her persistent low back pain and excruciating constant pain of the left lower extremity, without satisfactory result. Two years later, a high dorsal chordotomy was performed with partial temporary success. When she was 50 years old, that is, 16 years after her injury, I saw her for the first time. In addition to her intractable pain, she had a chronic psychoneurosis dating prior to her traumatic lesion, and a severe depression. A left frontal leukotomy made her comfortable for about one year, at which time a right frontal leukotomy was carried out, followed by several months of relief of her pain and improvement of her mental condition. The severe depression, psychoneurosis, and complaints of pain recurred to such a degree that they were unbearable to her and those around her. It was then decided to give cerebral intracranial EST as the last thing we had to offer. After a series of treatments, the marked clinical improvement of her emotional state and the absence of her pains were more dramatic than at any time following the leukotomy.

Comment

The above case offers an interesting demonstration of what may be accomplished with this method because of the results obtained in the patient's chronic mental syndrome, her severe depression and her additional problem of long-standing intractable pain. The clinical application of this method is intended for those conditions that do not respond to the usual electric shock therapy alone or combined with pharmacologic shock. Since it does not result in the extensive irreversible anatomic changes produced by psychosurgery it could, logically, be given a trial prior to these surgical procedures.

Discussion

Besides its therapeutic importance this new method opens new fields of investigation which may improve our knowledge of the functioning of the central nervous system and the etiology of mental illness.

Clinical usefulness of these methods can be found for those prob-

lems which do not respond to the usual electric shock therapy alone or combined with pharmacologic shock. Although it is a more radical type of treatment than the standard electric shock therapy, it would not result in the extensive irreversible anatomic changes produced by the common psychosurgical methods and should logically precede these surgical procedures.

In schizophrenics, when adequate series of electric shock alone or combined with insulin are failing, the aid of psychosurgery is sought. The greater the anxiety, the better the response to psychosurgery. The neurotic symptoms (obsessive, compulsive, tension, hypochondriasis, etc.) are the most susceptible to improvement. On the other hand, the depressive states which usually have a good prognosis after shock therapy, not only do not have a tendency to improvement but often increase following psychosurgery. Depersonalization favorably influenced by psychosurgery does not improve with the same reliability after shock. Our present problem in the field of schizophrenia will greatly be diminished by a type of treatment that would have the beneficial qualities of shock therapy and psychosurgery. In other words, if symptoms of anxiety, neurosis, and depersonalization would improve as with psychosurgery and, at the same time, if the depressive symptoms would respond as they do to shock therapy, the application of electric shock selectively and directly to the brain could be used to obtain the therapeutic effect of both shock therapy and psychosurgery and it might well be that insidious schizophrenia of long duration, which has such a poor prognosis with all methods actually used, would no longer be such a problem, or at least such a large problem as it is now.

Frequently psychotherapy fails in psychoneurosis. The poor results of electric shock therapy in such conditions is well known. That is why psychosurgery is sometimes recommended in chronic psychoneuroses which do not respond properly to psychotherapy, without having been submitted to preliminary electric shock therapy. Psychoneuroses due to the many problems not yet solved offer a particularly interesting field to clinical research. It is perhaps in this group that direct cerebral shock therapy may offer new possibilities. The same can be said of obsessive compulsive neuroses, since these conditions are the most chronic and the ones, of all the neuroses, that fail most often to respond to psychotherapy.

Although the effective disturbances respond almost specifically to electric shock, certain manic depressives, in whom the diseases persist for many years with short symptom-free periods, are submitted to psychosurgery. This is the last resource and is used in spite of reliable observations of authors that have pointed out several contraindications although many others are definitely opposed to surgical treatment.

Probably this is the ideal group to be submitted to this new procedure, since perhaps the good effect of shock therapy, well recognized in effective imbalance, could be improved in those patients in whom the usual external application has failed; in this manner, one might obtain such an improvement that the usual psychosurgical methods, of doubtful value in these patients, could be totally and definitely excluded.

The problem of pain which does not respond to the usual therapeutic methods could be briefly summed up as follows: Intractable pain has been treated by electric shock with variable results, and the unilateral or bilateral lobotomies are accepted as a specific treatment for intractable pain when chordotomies and other neurosurgical procedures have failed or are not indicated. It seems that lobotomy is effective, not because it interrupts the pain fibers, but because the perception of pain is blocked or changed in the patient's mind. In other words, we submit a patient who is normal mentally to an operation that will produce personality changes and risk having the same type of personality changes that we find in some mental patients, although probably of a lesser intensity. There is no doubt that such a risk, and even the personality changes, as severe as one often sees, are justified as a substitute for excruciating intractable pain even if, as it so often happens, the pain remains but its presence does not effect the patient and permits him a comfortable life without suffering. An ideal situation would exist if we could have a therapeutic procedure that would give more constantly and permanently the good result obtained sometimes in certain problems of pain with electric shock therapy and more frequently, with lobotomies, but without the personality changes that often appear in bilateral lobotomies. Perhaps this ideal therapeutic situation exists with the application of electric shock directly and selectively to the brain, and this possibility deserves more consideration and study.

No doubt this new technique may have its place in hypochondriasis, drug addiction, and chronic alcoholism.

Summary

A procedure to treat mental illness which allows a direct action of electric shock therapy over specific cortical or parenchymal brain areas is discussed.

A method for direct selective intracranial cerebral electric shock therapy is described. Since it will not result in the extensive irreversible anatomic changes that are produced by psychosurgical methods, it should logically be given a trial prior to the use of those surgical procedures.

1010 Fifth Avenue,
New York 28, N. Y.

SOCIETY PROCEEDINGS

NEW YORK NEUROLOGICAL SOCIETY AND THE NEW YORK ACADEMY OF MEDICINE, SECTION ON NEUROLOGY AND PSYCHIATRY

Joint Meeting, October 13, 1953

RICHARD M. BRICKNER, M.D., PRESIDENT, NEW YORK NEUROLOGICAL SOCIETY, *Presiding*

PRESIDENT'S ADDRESS: ANALYSIS OF THE NEURAL ORGANIZATION UNDERLYING MENTATION (DERIVED FROM A SYSTEMATIC STUDY OF THE PRODUCTIONS OF NARCOTIZED PATIENTS). RICHARD M. BRICKNER, M.D.

The verbal productions signifying mentation of individuals in amobarbital narcosis are just as systematized and just as readily subject to classification as any other activities of the central nervous system.

The drug acts on neural tissue, not on psychologic constructs like the ego, the super-ego, etc. The ensuing changes in mentation are the consequences of this pharmacologic action upon the central nervous system.

The "C-organization" is the name assigned to that portion of the brain organization, functioning as it does in amobarbital narcosis, which is responsible for the types of mentation described.

One of the chief functions of the C-organization is the fractioning of units of thought into smaller divisions, and the recombining of these fractions into highly varied combinations.

The psychologic and social roles of this function are discussed.

Discussion.—DR. PAUL H. HOCH: We are indebted to Dr. Brickner for these studies. He has confirmed and extended our knowledge of central nervous system reaction to amobarbital narcosis. He found that verbal productions in the patients are just as systematized and subject to classification as other activities of the nervous system. We believe this statement to be essentially correct, but a question of specificity arises. Are the manifestations described by Dr. Brickner observed only in amobarbital narcosis? No, this is not the case. They are seen in narcosis with other barbiturates—in some cases of ether anesthesia, under the influence of certain drugs, in the framework of an acute organic reaction caused by different agents, and also in certain forms of schizophrenia.

Dr. Brickner called attention to the fact that there are basic nervous system functions which can be understood independently from the motivational content. This is most important because there are those who are preoccupied with the investigation of motives, thereby leaving other functions untouched. But it is both the basic functional properties of the nervous

system and the more elaborate and symbolic ones together which give us a meaningful and whole story.

Dr. Brickner: I do not know how to reply to what Dr. Hoch said without going into still further great elaboration, which I won't do. I would like to mention my agreement with him concerning the similar effects of other narcotics.

High Cervical Cord Compression, Secondary to Rheumatoid Arthritis. Murray E. Margulies, M.D.

Spontaneous subluxation of the atlanto-axial joint has been described chiefly in children. Rarer instances are reported in cases of actual disease of the vertebrae or their ligamentous attachments. In view of this, it was decided to report this case. This is a colored male, right-handed, 35 years old, a hospital attendant, complaining of severe pain at the nape of the neck, radiating upward to the back of the head, inability to turn the head and to bend the spine, weakness of the right upper extremity, and difficulty with walking. His illness started in 1935 with stiffness of the muscles of the neck. The symptoms progressed slowly until September 1952, when he developed most of the above-mentioned symptoms. In November 1952, he was admitted to the hospital. Neurologic examination revealed primarily a Brown-Sequard syndrome, with motor paralysis on the right and sensory deficit on the left. Radiographic examination revealed a complete forward subluxation of the atlanto-axial joint. As the patient did not respond to conservative therapy, he was immobilized by means of a Minerva plaster jacket and subjected to a laminectomy. At operation it was found that the atlas was fused to the skull. Two exostoses were noted compressing the dural sac. There were carefully removed and following this two bone grafts were placed in such a position as to fuse the occiput and atlas with C_3 and C_4. The postoperative course was uneventful. The neurologic signs cleared up rapidly.

Titrud recently reported the first case in which an emergency cervical cord decompression was performed successfully in a patient who developed quadriplegia as a result of spontaneous atlanto-axial dislocation following a sore throat. The case presented tonight is the second instance of successful decompression following spontaneous atlanto-axial dislocation, secondary to rheumatoid arthritis.

Discussion.—Dr. Abraham M. Rabiner: To the neurologist this patient presented the problem of a deformed spine over which orthopedists were perplexed as to therapy and internists speculated as to etiology. However, after 18 years, this spondylosed spine now was associated with the clinical picture of high cervical cord compression. He had a modified Brown-Sequard syndrome and we concluded that surgical intervention was indicated. The technical difficulties experienced by the surgeons have been vividly presented by Dr. Margulies. The therapeutic result has been dramatically satisfactory to the patient and to all the physicians who participated in the study of the case.

Dr. E. Jefferson Browder: I presume when Dr. Margulies said that

this was the second case of spinal cord compression at this level that had been relieved by laminectomy he intended to imply that it was the second example secondary to rheumatoid arthritis. Dr. Margulies and his surgical colleagues are to be commended for the successful outcome in this particular example. Interestingly enough, there are a few cases in our experience of fractured dislocation or compression fracture of vertebrae at the level under discussion in which following the application of the Crutchfield tongs, a bad situation is not infrequently made worse. In one such situation last year at open operation there was no evidence to signify why this patient was made worse by traction on the head. It would seem in certain instances that an already sick cord, impaired by some alteration in its circulation, can be made worse by traction with the so-called Crutchfield tong.

Dr. Peter G. Denker: I would like to ask Dr. Margulies to explain one thing. According to the paper and its title, he seemed to lay all the stress on the marked spondylitis, but what produced the marked dislocation in the patient's neck? I think the subluxation was the crux of the matter in producing the cord compression at that level. Was there an accidental trauma somewhere along the line? Why so much stress on the arthritis which seemed almost coincidental?

Dr. Margulies: The arthritis was not coincidental. I inquired about trauma but the patient denied it. Apparently, just prior to the subluxation. the patient received penicillin and ACTH. I discussed the problem with an internist who felt that ACTH is not likely to be responsible for the subluxation. We have here, then, a patient with an atlanto-axial subluxation without any trauma or any cause that we could find other than the pathologic process in the ligaments of the vertebrae associated with rheumatoid arthritis.

Dr. Denker: Do you think the subluxation is related to the arthritis?

Dr. Margulies: I do.

Dr. Meyer Rosenberg: I think from the pathology found at operation in regard to the spontaneous fusion of the various vertebrae one can realize the subluxation was directly due to the arthritis. This man's entire spine was fused; all the vertebrae were fused, one to another, except at C_1 and C_2. The only place where spontaneous fusion had not occurred was between C_1 and C_2. With the normal motions and use of the head and bringing the head down gradually, naturally C_1 subluxated anteriorly, since that was the only place where fusion had not occurred.

Dr. Richard M. Brickner: I should like to ask about the distribution of pain, if any, particularly in the head.

Dr. Margulies: The pain was very severe, and within the radicular distribution of C_2 and C_3, over the back of the neck and head.

Clinical Evaluation of Mysoline, a New Anticonvulsant Drug. Daniel Sciarra, M.D., (by invitation), Sidney Carter, M.D., Carmine T. Vicale, M.D., and H. Houston Merritt, M.D.

A new compound, Mysoline, was administered to 121 patients with one or more types of convulsive seizures. Its anticonvulsant effect could be evaluated in 72 patients who were observed for periods ranging from 1 to 18

months. In all but 7, Mysoline was given in conjunction with one or more of the standard anticonvulsant drugs.

Seizures were completely controlled in 10 per cent, reduced in frequency in 43 per cent, and unchanged in 47 per cent. The greatest benefit occurred when the seizures were of the grand mal, psychomotor, minor, or focal types. No improvement was noted in any of the patients with petit mal.

Side-effects occurred in 65 patients but none were serious. Drowsiness and ataxia were the two most frequent symptoms and made it necessary to discontinue the administration of Mysoline in 25 patients.

Discussion.—Dr. Theodore J. C. von Storch: We have a series of some 50 cases, as yet not completely studied, the results of which are almost completely identical with those reported by Dr. Carter and his co-workers. We have only two differences in our series. A large percentage of our patients were eventually transferred completely to Mysoline, so that our results concern the effect of Mysoline alone. One other factor is that, although we have also had a high incidence of side-effects, by starting these same patients over with much lower dosages, we have in many of the patients been able to obviate the side-effects.

Dr. George H. Hyslop: What are the pharmacologic standards for comparing this new drug with phenobarital? It is so closely akin chemically, and obviously acts as a sedative, and with the large dose given, 1 Gm. equivalent to 15 gr. of phenobarbital by weight, I think if one is going to use this drug he ought to have something to judge it by other than some statistics on patients over one-third of whom you could not follow through.

Dr. Morris B. Bender: We also studied a series of patients treated with Mysoline at the Mount Sinai Hospital. Our results are the same as Dr. von Storch mentioned in his series. Our patients were mostly treated with Mysoline alone, rather than in combination with other anticonvulsant drugs. Moreover, the side-effects in our cases were not as marked as Dr. Carter described in his series, probably because the dosage we used was much lower than the dosages he used.

Dr. von Storch: I do not want to be misunderstood; the side-effects in our series were quite prominent and quite unpleasant and required cessation of the drug in many cases, but in some, by starting over at a lower dosage, we could treat them without severe side-effects.

Dr. Carter: We would like to make it clear that we do not have a new wonder compound. This is a drug that is effective in some 53 per cent of patients who are resistant to other anticonvulsant drugs. What this does in the patient who has never had medication before is hard to say. Why did we not discontinue other medication when we were using Mysoline? If we had a patient under control we did not quite have the courage to take away the other medication. Perhaps we did not have enough faith in the drug by itself. The toxic effects of this compound in this series are probably high because it was given in addition to other anticonvulsant drugs, and in many cases we could reduce the toxic effects by reducing the dosage of the other anti-epileptic medication.

BRAIN ALTERATIONS FOLLOWING EVACUATION OF INTRACRANIAL EXTRACEREBRAL HEMATOMA. ALBERT W. COOK, M.D., (BY INVITATION) AND E. JEFFERSON BROWDER, M.D.

Accumulated serial pneumoencephalograms in instances of intracranial extracerebral blood masses have been reviewed in an effort to obtain further information regarding brain distortion and swelling. Twenty-four patients were examined; 22 had subdural hematoma; in 3 the lesion was bilateral. There was one subdural hygroma and one epidural hematoma.

Generally speaking, persistent ventricular displacement endured for varying periods following evacuation of the extracerebral blood collections. The significant correlation of these cerebral alterations was that as the cerebral swelling regressed as measured ventriculographically, the clinical state improved. The degree of persistent ventricular distortion ranged from 0.5 to 2.0 cm., and in this series the longest time in which ventricular shift was observed was 52 days.

The radiographic evidence documents the persistence of these cerebral alterations during life and supplements the pathologic data relevant to focal cerebral swelling. In addition it dates these changes in relation to both the time and later evacuation of extracerebral compressive masses. The sequence of events in lesions such as subdural hematoma is as follows: in response to the presence of an extracerebral mass the homolateral cerebral hemisphere manifests varying degrees of focal swelling which persists and may even increase after removal of the hematoma. The correlation of these temporal factors with the clinical state lends further credence to the basic process of focal cerebral swelling as the causative factor for the neurologic abnormalities. In our opinion this is a more logical concept than those related to physiochemical alteration in the hematoma itself. These latter proposals of necessity leave unexplained the pathologic, radiologic, and clinical phenomena observed following the evacuation of extracerebral blood collections.

Discussion.—DR. BRONSON SANDS RAY (read by Dr. Masselink): The points made in this paper seem to me to be logical and certainly helpful in explaining many of the difficult aspects in these cases. In our use of radioactive tracers we have found invariably that there is a high uptake of the radioactive material on the side of a subdural hematoma. We know this is not due to the hematoma itself, because the fluid that has been evacuated has been tested for radioactivity and contains virtually none. The findings, however, indicate that there must be very significant changes taking place in the brain on the side of the hematoma which almost certainly would result in alteration in the blood-brain barrier. Under these conditions, the dye containing radioactive material would be taken up in significantly high amounts. We have not repeated the dye studies after evacuation of the hematoma, and this might be an interesting study to pursue.

DR. HERBERT PARSONS: I would like to ask if there is any additional correlation in the actual neurologic signs, such as dilated pupil, hemiparesis, and so forth, which go along with this change in cerebral edema. Perhaps

it would be a somewhat better explanation than the explanations which we have had previously.

Dr. Cook: In answer to Dr. Parsons' question, in all those patients who had postoperative examinations and who manifested varying degrees of persistent ventricular distortion, as the distortion regressed, the clinical features, namely, the hemiparesis and mental alterations, also reverted toward a more normal state.

Dr. Parsons: I gathered that, in general.

Dr. Cook: We made no specific correlation regarding dilatation of the pupils when it was present with cerebral swelling postoperatively. In the patients who manifested swelling, as recovery ensued, so did the swelling regress. There was only one patient in whom there was persistent stupor following evacuation of a hematoma. This man was reoperated on the eighth day after the demonstration of a shifted ventricular system, and no clot was found, only a swollen brain. After 30 days he had recovered, and his ventricular system by radiogram was back to normal.

BOOK NOTES AND REVIEWS

Books received are acknowledged and given a brief description in this section. Selections serving a variety of interests will be made from these for a more extensive review, as far as space will allow.

BOOK NOTES

Gellhorn, Ernest. PHYSIOLOGICAL FOUNDATIONS OF NEUROLOGY AND PSYCHIATRY. [Minneapolis: University of Minnesota Press, 1953. Pp. 556, with 107 illustrations. $8.50.]

This book presents the results of many years of investigation into the problems of nervous integration, a field in which the author is a recognized leader. The text includes and assesses what is known concerning the various factors of regulation of neuronal activity, the physiology and pathology of movements, the physiological basis of consciousness, several aspects of physiology of the autonomic nervous system, the principles of integration and the practical applications of this body of knowledge.

The text is fully documented, well illlustrated, and there is a bibliographic index of 1,263 references. The workers in laboratory, and clinical investigation will find it up to date with the fundamental advances that have been made in recent years in neuropharmacology, homeostasis, and neuroendocrine functions. It is highly recommended to all neuropsychiatrists and particularly to those engaged in research related to the somatic therapies in psychiatry.

Haymaker, W. (ed.), THE FOUNDERS OF NEUROLOGY. [Springfield, Ill.: Charles H. Thomas, 1953. Pp. 479. $10.50.]

This outstanding book consists of a compilation of one hundred thirty-three brief biographical sketches with photographs of the most important figures in the history of neurology during the past hundred years, written by eighty-four authors. Living persons are excluded. The sketches include mainly French, German, English and American investigators whose biographies are presented in groups according to the field of their particular contributions, namely, Neuroanatomy, Neurophysiology, Neuropathology, Clinical Neurology, and Neurosurgery.

Dr. Haymaker, his collaborators, and the Army Institute of Pathology and the publishers deserve great credit for this splendid service to students of Neurology. Those who have not read this book would do well to obtain a copy. It is interesting and informative in every part, and once started it will be read through without much delay. Well documented and indexed, it will serve for years to come as a reference

work. The reproductions and printing are excellent. It deserves to occupy a place on the active reference shelf of every teacher in the field.

Jones, Ernest. THE LIFE AND WORK OF SIGMUND FREUD, Vol. I., THE FORMATIVE YEARS AND THE GREAT DISCOVERIES, 1856-1900. [New York: Basic Books, 1953. Pp. XIV 428, illustrated. $6.75.]

Psychiatrists and educators throughout the world will be intensely interested in this masterly biography of the originator of psychoanalysis. No other person now living could have written so authoritatively of the life of Freud than Jones, who was for many years a disciple, personal friend and co-worker in the development of psychoanalytic theories and proceedings of the great investigator of the unconscious processes.

The biography is to be published in three volumes. Volume I describes the first forty-four years of Freud's life, presenting the facts, including new material, concerning early childhood, boyhood, adolescence, early professional training and experiences, courtship, marriage, and economic struggles and finally the orientation, the penetrating studies along the way, and the fruitful results of the persistent efforts and deep convictions that characterized Freud's progress in making his remarkable discoveries.

There are seventeen chapters in the text, the final one presents Freud's theory of the mind to the point he had developed it in 1900. The work is well documented and indexed, is rich in style and anecdote and is unique in its coverage. Dr. Jones has rendered a great service to the many disciplines concerned with psychoanalytic history, theory and practice, and one looks forward with keen anticipation to the future volumes completing the story.

Mitchell, G. A. G. ANATOMY OF THE AUTONOMIC NERVOUS SYSTEM. [Edinburgh and London: E. S. Livingstone Ltd. and Baltimore: Williams and Wilkins Co., 1953. Pp. 356 with 131 illustrations $10.00.]

This new text on the anatomy of the autonomic nervous system is a masterpiece of accuracy and exposition. The author knows how to write a textbook of neuroanatomy in a way refreshing and unique in that he is able to show clearly how the sympathetic and parasympathetic divisions of the autonomic structures are integrated with the central and peripheral nervous systems.

Another feature adds to the interest of the research worker, namely an attitude of the author as stated in his preface, "the views expressed herein are occasionally unorthodox and sometimes provocative, but some conceptions, founded on slender evidence, have been too readily accepted and repeated. A few pebbles cast into the placid pools of complacency readily display the doubts and difficulties lurking in the depths and their revelation is the first step toward their solution."

There are eighteen chapters of text, well documented and illustrated

by unusually clear illustrations, many in color, a useful bibliography, and a complete index. These features, together with the excellent print and binding of the book, will contribute to its usefulness to anatomists, physiologists, neurologists and neurosurgeons.

Walker, A. Earl, (ed.). A History of Neurological Surgery. [Baltimore: The Williams & Wilkins Co., Pp. XII 583 Pp. 152 illustrations. $12.00.]

This volume is both a remarkable historical account of neurosurgery and a splendid piece of bookmaking. Dr. Walker and his group of assistants in the Johns Hopkins Clinic have produced a book that will live for years to serve teachers and students in this field with a background and a tool for progress.

The illustrations are of uniformly high quality, both as to their selection and reproduction, and each chapter with the third is followed by an interesting biographical sketch of some noted figure in the development of neurosurgery.

Many neurosurgical problems are described among which are congenital anomolies, epilepsy craniocerebral trauma, cerebral vascular conditions, epilepsy spinal cord surgery, surgery of the peripheral nerves and a chapter on the autonomic nervous system.

This scholarly volume will be invaluable as a reference source since in addition to the topics discussed, there is a bibliographic list of 2,371 items affording a sweeping survey of the whole field of neurosurgery as it has developed internationally.

Wechsler, Israel S. A Textbook of Clinical Neurology, 7th Ed., [Philadelphia: W. B. Saunders Company, 1952. Pp. XIV 801. $9.50.]

The seventh edition of one of America's basic neurological texts represents a comprehensive revision of the previous edition published in 1947. The author stresses the fact that while the volume preserves its pattern, hardly a page has been left unedited, illustrations have been added and replaced, complete revision of many of the treatment sections has been accomplished, classification techniques have been brought up-to-date, and new material incorporated in many areas. These changes modernize a most workable volume which is of particular value to the student requiring basic information, as well as for the expert needing a refresher survey. This text has always been one of the easiest to read, and can be considered to cover the material necessary without becoming lost in detail.

The author has also included an excellent introduction to the history of neurology, and, as always, has a separate part dealing with the neuroses. Just why the history of neurology is tucked away in this part, however, is not quite clear.

In the current edition Wechsler stresses the fact that he has "critically reviewed the chapter on the neuroses, re-defined certain concepts,

and eliminated older ones." This revision is certainly valuable, but the superficial descriptions necessitated by the minimal space allotment yield little real information. Statements like "many (sexual) perversions are to be viewed as psychotic symptoms" are typical of the loose thinking presented, and a careful reading of this section impresses one that it is more of a glossary than a discussion.

It can certainly be said that this text is an outstanding one from which the psychiatrist can learn neurology and, on the other hand, a very poor place for the neurologist to learn psychiatry.

[Douglas M. Kelley]

Werner August A., and Associates. RESEARCH IN ENDOCRINOLOGY. [St. Louis: Von Hoffmann Press, Inc., 1952. Pp. 285.]

This book is apparently a compilation as a tribute to Dr. Werner. The text consists of five chapters, the first four of which include biographical data, lists of publications, some of his lectures and abstracts of his numerous researches. The last chapter comprising three fourths of the book presents sixteen reprints of articles published in the period 1931 to 1946. These deal chiefly with the effects of estrogens and testosterone when used therapeutically. A relationship between involutional melancholia in women and ovarian hypofunction is postulated. It brings up some interesting problems in endocrine physiology.

NOTES AND NEWS

ANNOUNCEMENT

The University of Minnesota announces a continuation course in Psychiatric Principles in General Practice from September 8 to 10, 1954. The course will be held at Douglas Lodge, located deep in the woods of northern Minnesota on the shores of beautiful Lake Itasca. The program will concern those psychiatric and psychosomatic problems which are most frequently seen in general practice, and practical management will be emphasized throughout. The guest faculty will include Dr. Bernard C. Glueck, Jr., Ossining, New York, and the course will be presented under the direction of Dr. Donald W. Hastings, Professor and Head, Department of Psychiatry and Neurology, and Director, Division of Psychiatry. The remainder of the faculty will be drawn from the University of Minnesota Medical School and the Mayo Foundation.

Sessions will end each day in mid-afternoon to permit time for fishing, boating, and other recreational activities. The fee will be $130.00 which will include registration, tuition, meals and lodging, instructional material, transportation to Douglas Lodge and return, and fishing license. Further information may be obtained from the Director of Continuation Medical Education, University of Minnesota Hospitals, Minneapolis 14, Minnesota.

St. Louis, May 6—Hon. John Biggs, Jr., Chief Judge of the U. S. Court of Appeals (Third Circuit), Wilmington, Delaware, has been given the American Psychiatric Association's $1,000.00 "Isaac Ray Award" for 1954 for his notable contributions to the field of legal problems connected with mental disorders. The Award was announced at the Association's annual meeting at St. Louis, May 5th.

As recipient of the Award, Judge Biggs will deliver a series of six lectures on legal aspects of psychiatry in November and December this year at the University of California Medical and Law Schools. The lectures will then be published in book form by the Harcourt Brace Co., publishers.

In conferring the Award, Dr. Frank J. Curran, Professor of Psychiatry at the University of Virginia School of Medicine, Charlottesville, said that Judge Biggs was in the vanguard of the legal profession in his efforts to make courtroom procedure accord with modern knowledge of psychiatry and the dynamics of human behavior.

The Isaac Ray Award is given annually to a lawyer or psychiatrist in the interest of furthering understanding between the two professions which are so often mutually concerned in legal questions involving mental disorder.

The Award is commemorative of Dr. Isaac Ray, a founder and early President of the APA, whose "Treatise on the Jurisprudence of Insanity" (1838) was a pioneering work in this field. The winner of the Award is asked to deliver from three to six lectures at a university which incorporates both medical and law schools. Judge Biggs is the third winner, Dr. Gregory Zilboorg, noted New York City psychiatrist, and Dr. Winfred Overholser, Superintendent of St. Elizabeths Hospital, Washington, D. C., being the other two.

Judge Biggs was selected by a board comprised of Drs. Curran, Zilboorg, and Overholser, and Drs. Francis J. Braceland, Hartford, Conn.; Leroy A. Maeder, Philadelphia, Pa.; Paul Schroeder, Atlanta, Georgia, and George S. Stevenson, New York City; and lawyers Ralph C. Busser, Philadelphia, Pa., and Walter A. Edwards, Providence, Rhode Island.

INSTRUCTIONS TO AUTHORS

1. Bibliographies should be in alphabetical order. These references should include: name of author, title of article, name of journal, volume, page and year. When books are cited, include author, title, year of publication and publisher.

2. Footnotes should be omitted unless very brief. Material usually placed in footnotes should be incorporated in the text.

3. Manuscripts, which should represent the final form of the material, should be typewritten double spaced, including footnotes, case reports, bibliographies, and only the original will be accepted for publication. Handwritten corrections must be legible and kept to a minimum.

4. Illustrations for halftones should be glossy photographic prints; for line engravings, drawings should be made with black India ink on white paper or blue graph paper.

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